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CONCERNING SURGICAL INTERVENTION FOR THE INTRACRANIAL HEMORRHAGES OF THE NEW-BORN.[1]

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In a recent paper[2] some generalizations were made in regard to the propriety of operative measures for certain maladies, primarily of neurological interest, that are commonly supposed to be beyond therapeutic aid. The cerebral palsies of children were among the conditions then cursorily reviewed, and the belief was expressed, that, at the time of onset, the indications for immediate surgical intervention are as definite in many of these cases as they are when corresponding symptoms are unmistakably the result of a traumatic intracranial hemorrhage occurring in adult life. This expression of opinion has called forth so many inquiries and led to so many friendly comments—not always of approval, it must be said—that I have been persuaded to amplify the original paragraphs and to record in greater detail my few operative experiences in order to make my position in the matter more clear.

For the convenience of clinical grouping, a variety of terms are used to indicate those individuals who give evidence of serious cerebral defects that have been acquired during infancy. In the main, these terms are descriptive merely of a common residual symptom—the unilateral or bilateral spastic palsy—and this means nothing more than that the so-called motor strip of the cortex, to a greater or lesser extent, on one or both sides, has suffered irreparable injury early in life from an intracranial lesion. Although

[1] Presented at the Philadelphia meeting of the American Neurological Association, June 2, 1905.

[2] The Special Field of Neurological Surgery, Johns Hopkins Bulletin, April, 1905.

it must be kept in mind that the spastic paralysis is by no means a necessary consequence of the lesion—for the cortical injury may be limited to an area of the brain remote from the central gyri, and so lead to no motor disability whatever—the integrity of the cortico-spinal efferent path is, as a matter of fact, and for reasons to be given, very apt to be implicated. A small lesion, which in any other situation might pass unobserved, when placed here will later in life give unmistakable evidences of its presence, as is well shown by a case to be mentioned below (Fig. 1). In the more serious injuries both hemispheres may suffer, and if the lesion is widespread, not only may the spastic diplegia, so well depicted by Little in the middle of the past century, be a consequence, but also varying grades of amentia as well.[1]

Inasmuch as most of the cases are not immediately fatal, being serious only from the distressing late consequences of the injury, there is little known of the morbid anatomy of the disease in its inception. It is, however, generally conceded by the many writers on the subject that a cerebral vascular lesion—hemorrhage in a large majority of cases—is the causal factor of the malady of prime importance. This applies to the cases of prenatal as well as to those of postnatal origin, but more particularly to those incident to parturition—the so-called "birth palsies," with which this paper primarily deals. In these the intracranial hemorrhage is usually of venous origin and follows the rupture of some of the delicate and poorly supported venous radicles of the cerebral cortex. Such an injury may be the direct result of undue traumatism during labor or may occur when too great strain has been put upon the vessels by the profound venous stasis of postpartum asphyxiation; just as in later months they may rupture under the passive congestion brought about by a paroxysm of whooping-cough or a severe convulsion. Though Little's three papers, doubtless more than the writings of any of his predecessors, served to call attention to the malady, he had very few pathological data with which to support

[1] It is, of course, presumable that there have been included in this large group of cases several diseases, which, from an etiological standpoint, are quite distinct, but which, being correspondingly destructive to cortical function, leave traces that in later years make them symptomatically indistinguishable. For example, should Strümpell's opinion that there is a cerebral infectious process—polioencephalitis akin to the more definite poliomyelitis—be finally established, a large group of cases, particularly those of the infantile hemiplegic type, could definitely be separated. Many have already accepted his views. On one occasion I performed a craniotomy on a child four years of age in an acute febrile attack, suffering with headache, vomiting, unilateral convulsions, and presenting a bilateral and rapidly forming stasis papilla. The trephination was conducted merely for the relief of tension, but with the hope at the same time that the symptoms might be due to a cortical hemorrhage. The exposed hemisphere was found tense, œdematous, and with a multitude of punctate hemorrhages scattered over the various convolutions. I presume that it was an instance of Strümpell's form of encephalitis, and on another occasion I should not hesitate to remove a small piece of the cortex for histological examination. The pathology of these conditions may more certainly be learned by early operative intervention than in any other way.

his contention, based largely upon clinical histories, that calamities of childbirth were responsible for a large proportion of the crippled and spastic children that had come under his care. This is the more apparent when one realizes how much importance has always been given by writers in all languages to the careful pathological reports which accompanied the papers of Dr. Sarah J. McNutt, published twenty-five years after Little's contributions called attention to the subject. Why such reports have been uncommon is not easy to explain, unless it is that autopsies of infants who have died seemingly from asphyxiation have rarely been conducted with this particular point in view. A few years ago, when interested in making some observations on the circulation of the cerebrospinal fluid, I was privileged by the obstetrical department to study, for the purposes of that investigation, quite a number of infants who had been stillborn or who had died during the early hours or days of life, and found, to my surprise, that a considerable percentage of them had died with an intracranial hemorrhage. This, too, has been the experience, I believe, of most pathologists, though records of such findings are astonishingly rare. Whether these cortical hemorrhages in the cases which I examined had been the cause of death could not, of course, always be told, though in some of them the effusion of blood was so great that little doubt was felt but that it must have played an important part in the asphyxiation which the clinical histories recorded. In one of the cases a large, thick clot was found overlying an entire hemisphere, and practically confined to one side, a condition which has been present in two of the cases on which I have since operated. In other instances the extravasations were smaller. One of them was in the cerebellar fossa, associated with a fissure in the occipital bone. In another case the chief distribution of the clot was over one of the occipital lobes.

When one considers what an immense amount of traumatism the infant's head must necessarily be subjected to, particularly should the child be large and the firstborn of an elderly mother, it is little wonder that lesions of this kind occur. And this even without the additional strain that may be occasioned by the instrumental delivery which a narrow pelvis or inelastic soft tissues may ultimately necessitate. The infant's brain itself is doubtless fitted by nature to withstand, without injury or shock, a degree of moulding and manipulation which could not be borne by an adult, fully developed, and medullated brain. And furthermore in cases of traumatic œdema, or of actual hemorrhage, the distensibility of the partially membranous skull will accommodate effusions of such extent that were they to take place in the closed adult skull in proportionate degree, they would rapidly prove fatal. It has been my experience that of the adult cases with an equally extensive hemorrhage only those recover in which there is an associated great fragmentation of the cranial vault, and this, by allowing for some cranial

expansion, releases the brain in a measure from the compression effects that otherwise would be produced. It is probably this distensibility of fontanelle and skull which lets many of these infants escape death, for, being at this time little more than spinal animals, they may live with their bulbospinal axis alone, even in case the cerebrum is thrown in large part out of function. The early days of life, however, when medullation is rapidly taking place, are, so far as cerebral development goes, possibly the most important of all, and it is natural to suppose that the pressure of a clot which may be long in absorbing, or which organizes slowly, will, through a local compression anæmia, prevent the normal development and lead to the cerebral abnormalities that we recognize as the late evidences of the lesion.

It is an impression gained from my own studies, and, I believe, one that has been advanced by others, that the vessels most likely to rupture are those which ascend over the cortex and enter the longitudinal sinus from the midcerebral region. These veins, which lie in the subarachnoid spaces, and for the most part are protected from pressure by following the sulci as they ascend toward the midline, leave this protection when near the vertex, and apparently with no support whatever cross the subdural space to enter the dura at the outer edge of the sinus. In the adult brain there are many adhesions between these cortical vessels and the adjacent dura, particularly where this underlies the lateral expansions of the longitudinal sinus, for here the membrane is punctured in many places by the granulations of Pacchioni. In infants, however, as is also the case in the lower animals, there are no such adhesions, the unsupported vessels being the only connection between the leptomeninges of the hemispheres and the dural sheath underlying the sinus. This can easily be seen by removing the infant brain and calvarium in one piece and by allowing the former to sag away from its bony and membranous covering.

During parturition there is presumably always some overlapping of the parietal bones, owing to the lateral compression of the skull, a condition thought to be more marked with the after-coming head or in the precipitous expulsion of a premature child, whose cranium is less resistant to pressure It is a condition, also, which must necessarily be accentuated by the lateral application of forceps.

Although the cranial moulding may take place to a considerable extent without putting any great strain on these lateral cerebral veins, any undue overlapping could readily tear one or more of them away from the sinus. This, I think, is the more common form of injury[1] and possibly accounts for the fact that the primary

[1] In two of the cases that I have examined I have satisfied myself that such ruptures were present. A positive statement, however, cannot be given even for these cases, since the dissection and exposure, difficult enough under any circumstances, owing to the delicacy of the vessels is the more so when they are obscured by extravasated blood. One of them was Case IV. of my series. Cases are on record in which the longitudinal sinus itself has been lacerated

effusion and so the thicker portion of the clot is commonly median, and, in consequence, implicates primarily and most seriously the centres for the lower extremities. The rupture of the vessels may take place only on one side, and I am of the opinion that the extravasation is usually limited to one side of the falx, though, in deed, a bilateral lesion is common enough, as the many patients with diplegia would indicate. Intracranial bleeding may, of course, result from lesions other than the laceration of veins from overlapping. Fractures, particularly basal ones, may be followed by a serious hemorrhage, and in these instances, as Case III. illustrates, the extravasation may not find its way to any great extent on to the outer surface of the hemispheres, but may infiltrate and affect primarily the basal ganglia. It is possible that injuries of this sort, particularly when the thalamus is involved, may be the ones which lead to the extraordinary condition of double athetosis; for in these cases so far as my experience goes—I have seen but one of them at operation—the hemispheres are absolutely normal in appearance, and it is noteworthy, as well, that in them epilepsy is a rare sequence and intelligence fairly well preserved.

In the numerous and valuable monographs that deal with the cerebral palsies of children—and particularly is it true of the birth cases—there is a remarkable absence of any description of the early symptomatology of the lesion; its consequences alone are given in detail. This may possibly be due to the fact that there is a silent period, so to speak, in the malady, during which the child "improves," and after some months, when it becomes evident that normal cerebral development is not taking place, the patient has very commonly passed out of the obstetrician's hands. So that beyond the account of the abnormal labor there are few data from which we may construct the symptomatology of the early intracranial lesion.

Judging from the patients whom I have seen, however, the condition need not be difficult of diagnosis. Had there been in any of the cases, a serious doubt as to the actual nature of the lesion, it might have been decided by a lumbar puncture, for when there has been any effusion of blood into the subdural spaces, free corpuscles will soon after be found in the fluid of the lumbar meninges. This has proven of great value in determining the presence of subdural bleedings in adult cases, and in infants might serve to differentiate between the hemorrhagic and infectious cases were diagnostic difficulties present. The history of the labor is, of course, all-important; also the degree of postpartum asphyxiation. Among the objective signs should be placed first and foremost the condition of the fontanelle. The membrane is bulging and in some instances may be so tight as to show no pulsation. In two of the patients its bony outlines, in consequence of the tension, could not be palpated,

and it has several times been learned from parents, whose hemiplegic children have been seen for the first time some years after birth, that the fontanelle could not be detected during the first days or weeks of life. Convulsions, of course, are frequent, and particularly if they do not appear until several days after birth they may be more or less unilateral in character. There may be also an undue reflex activity, the child starting or twitching in a convulsive way in response to any sensory stimulus. Ocular palsies or differences in the size of the pupils apparently are not uncommon, and in severe cases there may be symptoms referable to the medulla, as alterations from the normal in the cardiac or respiratory rhythms. In the early days evidences of paralysis are rare. There may be no difference observable in the muscular tone on the two sides of the body, even when the hemorrhage is confined to one hemisphere.

Thus far merely the initial lesion and the symptoms characterizing the onset of the disease have been briefly considered, and, though the critical stage of the malady, it is, to any but the obstetrician, its least familiar period. In its fully developed and terminal aspects the disease is better known—far too well known to neurologists, orthopedists, or attendants upon the asylums for feeble-minded children. A supposed restoration to health may gradually follow the first few precarious days of life, and not until some months have elapsed do the late manifestations of the disease begin to unfold and to bring to light the spastic palsies, or blindness, or deafness, or feeble-mindedness, or, in severe cases, even complete amentia.

There is one very serious sequel which frequently (two-thirds of the cases according to Gŏwers) accompanies the mildest grades of the disease, even when intelligence may have in nowise suffered. This is epilepsy; and the convulsions, often Jacksonian in character, are akin in all respects to those which follow the cortical changes resulting from cranial injuries with hemorrhage in adult life, and the late evidences of the lesion may have much the same gross appearance.[1]

[1] It may be said, too, in passing, that these cases of epilepsy associated with spastic palsies in children are almost as likely to be bettered by properly conducted craniotomies as are the post-traumatic epilepsies in the adult. During the past few years I have explored about thirty of these cases, and the abnormal conditions encountered defy description. I have seen a right-sided porencephalic cyst with spastic hemiplegia and contractures in a patient whose intelligence was practically normal; a microcephalic child with complete amentia, in whom both hemispheres were exposed, disclosing nothing that could deserve the name of a convolution. In some instances the dura and the cortex may be free, in others densely adherent, and at times there may be large, new-formed, venous sinuses over the lateral surfaces of the dura. In one such case a vertically placed sinus overlay the central area and was lined by Pacchionian granulations. There may be large areas of convolutional atrophy, and the situation of these areas is usually shown before the operation by configurative alterations in the shape of the skull. Twice I have seen in epileptics a flattening of this sort over one occipital lobe: they were cases of birth hemorrhage with homonymous hemianopsia. One of them was operated upon, disclosing a sclerosed and atrophied lobe with adhesions. There may, however, in these patients be no adhesions whatsoever between brain and dura, but merely some thickening of

It is this complication which brings many of these patients to the surgeon and gives him an opportunity of seeing at first hand and during life the extraordinary variety of the cerebral lesions that result from the original hemorrhage. In the adult, however, such a destructive cerebral lesion as may take place in infancy and still be compatible with life could hardly occur, and it is possible that the more firm and resistant character of the fully medullated brain may tend to check a venous extravasation which in the infant might have become huge and perhaps have been the forerunner of porencephaly.

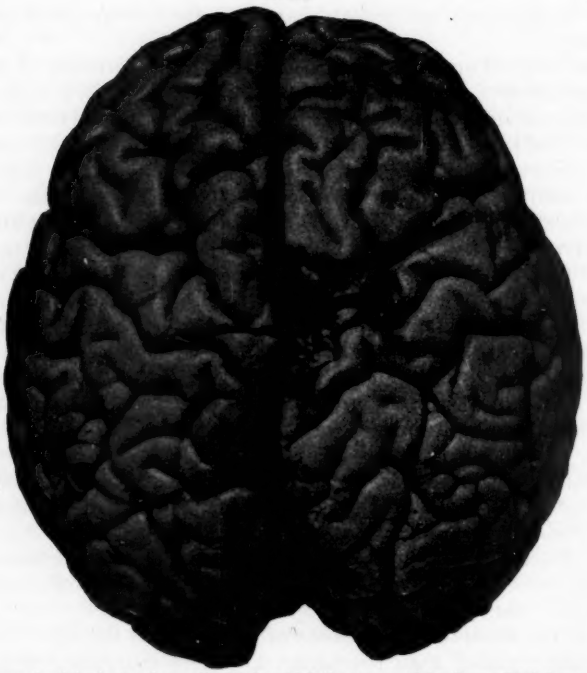
The brain of one case, which I have had an opportunity of studying postmortem, is deserving of mention in some detail, as it shows a lesion, resulting from a birth hemorrhage, which is limited to the motor area of the lower extremity on one side. The patient was a colored girl, thirteen years of age, the firstborn, who had been delivered with instrumental aid. Convulsions had occurred soon after birth, and she had suffered from epilepsy from infancy. The attacks were typically Jacksonian in character and always began in the left leg, which was spastic and much smaller than the other in all of its measurements. The child seemed normally bright and intelligent. She died in the hospital of an intercurrent trouble while awaiting operation. An area of sclerotic atrophy, as shown in the accompanying photograph (Fig. 1), was found, limited to the motor area of the left leg. It is here that lesions would naturally be expected to occur as a result of a rupture of the veins entering the sinus. A similar lesion, if bilateral, would have led to the paraplegia so commonly seen, or to the diplegia of Little if the hemorrhage had been extensive enough to reach to the motor centres of the arms. The remarkable way in which the cortical lesion may be confined to the Rolandic convolutions on the two sides is well demonstrated by the careful study, made in 1885, by Dr. Welch, of Sarah J. McNutt's case.

It is the absolute hopelessness of any form of therapy in these late stages—other than tenotomies to relieve the spastic contractures, or the surgical attempts to check the epileptic seizures—that have led to the despairing words with which the best-known writers on the subject dismiss the therapeutic section of their papers. In his valuable monograph Freud gives his own and a distinguished American neurologist's opinion as follows: "Die Therapie der In-

the pia-arachnoid, with an increase in the amount of fluid filling the sulci. In one patient seen with Dr. Osler and Dr. Thomas, of double athetosis associated with extremely painful cramps, both hemispheres were exposed at different sittings, and the cortex on each side was found to be perfectly normal in appearance, and to give clean-cut responses upon faradization of the precentral convolution. A large portion of this convolution was removed from one side, with a cessation of the cramps, but without affecting in any way the athetoid movements, and giving merely the usual temporary palsy of the extremity. Many other instances might be cited. In the whole series there have been no fatalities. The outer wall of the tense porencephalic cyst in two cases has been removed without appreciable symptoms. Dr. Starr, however, has recorded a sudden fatality following such a procedure.

fantilen Cerebrällahmung ist ein armseliges und trostloses Capitel sowohl an sich als im Vergleiche zu dem mächtigen klinischen Interesse, welches diese Affectionen erregen. Hier gilt, leider unwidersprochen, der Ausspruch von Allen Starr: 'Hemiplegia, sensory defects, and imbecility, occurring with or without epilepsy in children, are chronic diseases incurable by medical treatment.'"

FIG. 1



Photograph of the brain of an epileptic, in which the scar resulting from a birth hemorrhage is seen occupying the upper end of the right precentral gyrus (motor area of the left lower extremity). The entire convolution is smaller than the corresponding one on the left. Symmetrical incisions, due to removal of tissue for histological study.

It is hoped that in some instances, just as after serious cranial traumatism in the adult, the late results of the meningeal hemorrhage in these cases of so-called birth palsies may be warded off by an immediately conducted operation.

My four operative experiences are represented by the following cases:

CASE I.—The patient, a male child three days old, was seen in consultation with Dr. Dabney on the afternoon of August 22, 1903.

The child was the first of twins, born at full term of a primipara thirty-five years of age. Though a normal head presentation, the labor was slow and prolonged, and low forceps were finally applied. The delivery was easily accomplished without the exertion of force. The child was deeply asphyxiated and it was some time before it could be made to take a breath. Its twin brother, born a few moments later, was normal in every respect. The first child did very badly for the first two days. It could not suckle, and would only occasionally swallow the drops of milk which were put in its mouth. It had a peculiar dusky, cyanosed appearance. There had been several convulsive seizures.

The symptoms were the more obvious, as we had the normal child for a control. The fontanelle was tense and without pulsation. The left eye was kept closed and the pupil on this side was dilated, being twice the diameter of that on the right side. Both reacted to light, the right more than the left. The winking reflex was present on both sides, but only when the lashes were touched. The supraorbital reflex was present, more active on the left. The child could move its arms and legs, and there seemed to be no spasticity nor difference in muscular tone on the two sides of the body. The convulsions had not been unilateral.

Contrasted with its twin, whose pulse was 110 and regular, this child's pulse was considerably slower, having an irregular action of about 92 beats per minute. The respiration, which had been rather irregular and shallow from the beginning, had on this last day assumed something of a Cheyne-Stokes type of rhythm, with periods of apnœa. The child was prepared for immediate operation.

Operation. August 22d, 6 P.M. After the head was shaved and prepared, a few drops of chloroform were administered and a horse-shoe-shaped incision was made through the skin just within and concentric with the border of the left parietal bone. An osteoplastic flap was then raised, including almost the entire parietal bone. The bone was cut through with a stout pair of blunt, curved scissors, very close to its thin and serrated edge. On elevating the bone-flap a tense, dark, plum-colored dura was brought into view. When this membrane was opened and reflected, a clot about 1 cm. in thickness, apparently covering the entire hemisphere, was disclosed. This clot was lifted off in large pieces from the exposed area of the cortex with a blunt instrument, and a large amount of clotted blood which extended over the frontal and occipital regions was irrigated out from under the dura with warm saline solution. The wound was closed without drainage.

Despite its enfeebled condition, for it had taken practically no nourishment during its three days of life, the infant stood the operation remarkably well. A small subcutaneous infusion of salt solution was given, and two hours after the operation the child took some

nourishment from a bottle by sucking the nipple for the first time since its birth. Though there seemed every prospect of a recovery, the infant suddenly died during the night, about eight hours after the operation.

No examination was allowed, and though it would have been interesting to have learned whether there was an extravasation over the opposite hemisphere, it seems quite likely, in the light of the last case in this series, and in view of the unilateral palsy of the third nerve, that an effusion, even were one present on the right side, was less extensive than the one which had been disclosed at the operation.

CASE II.—The patient was first seen March 13, 1904. It was born of a primiparous parent a week previously, on the 6th of March. It had been a tedious breech presentation, with manual delivery. The head had been released with some difficulty. A condition of "pallid asphyxia" was present at birth, but in a few moments, after a hot and cold plunge, the child cried vigorously. There was no overlapping of the cranial bones or other obvious effects of traumatism. During the following night the child kept both of its eyes open most of the time, and for the next five days nothing unusual was noted, except this apparent wakefulness and some stupidity in nursing. The child was not fretful, its cry was strong, and it took nourishment from a bottle fairly well. It had begun to gain slightly in weight. On the fifth day there was a slight elevation in temperature (101.6°), for which there was no obvious cause. The pulse was somewhat slower than usual, registering between 80 and 100 beats per minute.

On the sixth day, Saturday, March 12th, at 9.15 A.M., convulsions first set in, the first spasm lasting perhaps two minutes. During the day there were five other convulsive seizures of varying type and of increasing severity. In the more severe seizures the most decided twitching occurred in the right arm and leg. Some of the convulsions were confined to the face and head, the arms and legs being quiet. In one of the attacks the eyes were crossed, with smacking of the jaws, and with the head thrown to the left, and some rigidity of the neck. The child would sleep after the convulsions, occasionally with its left eye tightly closed and the right partly open. At 11 P.M. there were several severe and repeated convulsions, with some opisthotonos and with a sharp cry at the termination of each.

On Sunday, March 13th, there were eleven convulsive attacks at varying intervals, several of which I observed, when late in the day the child was seen in consultation with Dr. E. F. Cushing and Dr. Crile, of Cleveland. Most of the seizures were bilateral in character, though some of them seemed to us more marked on the left side. There was a constant inequality of the pupils, the left being somewhat dilated, or the right contracted, except during an

attack, when both of them were wide. The fontanelle was not as tense as in the previous case, and, though slightly bulging, its bony outline could be made out.

Monday, March 14th. Up to 9.30 A.M., an hour before the operation, there were seven convulsive seizures, more severe than on previous occasions, and associated with occasional cyanosis. They were for the most part left-sided, and were accompanied with a conjugate deviation of the eyes to the left. One of the spasms lasted five minutes. After the attacks a clear, non-frothy saliva with mucus would pour in considerable quantities from the child's mouth.

The operation, at which I was assisted by Dr. Lower, was performed at 10.30 A.M. Every effort was made to preserve the warmth of the patient and she was wrapped thickly in cotton. The head was shaved, and, as in the case above described, under chloroform anæsthesia, the parietal bone was turned down with its overlying soft parts, this time on the right side. The dura was found to be somewhat tense, and though not deeply plum-colored, as when it covers an extensive clot, it nevertheless showed the presence of an underlying extravasation. The membrane was opened, turned down, and the free blood and small separate clots which were scattered over the surface were irrigated away with warm salt solution. The wound was closed without drainage.

The child slept for two hours after the operation, awoke, cried vigorously, and took its nourishment eagerly. The convalescence was without incident. The wound healed by primary union, the slight pyrexia, which had been present for some days, subsided, and at no time following the operation was there a suggestion of any further convulsive seizures.

On March 28, 1905, the attendant writes me: "The child was a year old March 6th; it stands, takes a few steps, says a word or two, and is a fine specimen of wholesome babyhood."

CASE III.—The patient, with the following history, was seen for the first time October 11, 1904, in consultation with Dr. J. Whitridge Williams. A large baby, a girl, was born October 3d, after a difficult labor lasting forty-eight hours, which was finally terminated by a low forceps operation. At birth the child was profoundly asphyxiated, and only after vigorous efforts was spontaneous respiration established. There was a small abrasion, evidently made by the instruments, over the frontal region. This little wound bled considerably, healed up partially, and later on began to bleed again, so that it was thought the child might possibly be hæmophilic. On the day following birth the right eyelids began to swell somewhat, and there was considerable œdema in the surrounding tissues. On the third day there was an extreme protrusion of the eyeball (Fig. 2), which appeared in the course of a few hours. For several days after birth the child nursed fairly well at

the breast, but later on it refused or was unable to suckle, and nursing on October 8th had to be discontinued. The child became dull and stupid and would not swallow even the nourishment put in its mouth. Its respirations were very irregular. On the morning of October 10th they became markedly Cheyne-Stokes in character, and in the afternoon there was great restlessness and convulsive movements of the arms and legs. Twitching, particularly of the right hand, was noted, and some rigidity of the legs was first observed on this date. The nurse had noted that the child's pulse often registered below 100 beats per minute. The fontanelle was exceedingly tense.

FIG. 2

CASE III.—Photograph of the nine-day-old comatose infant, taken the morning of operation. Extreme degree of ocular proptosis, due to cavernous sinus thrombosis.

When first seen by the writer it hardly seemed possible that the child could survive, and indeed its life had been for a day or two despaired of. The most startling feature was the extraordinary protrusion of the right eyeball with corneal opacity and hemorrhagic œdema of the lids and conjunctiva. The fontanelle was bulging and so tense that its bony margins could not be made out; there was no pulsation. The radial pulse was very irregular in rhythm and quality. The respiration was irregular, with long periods of apnœa.

There seemed little question as to the presence of an intracranial hemorrhage, though considerable doubt existed as to its exact situation. The history of a sudden non-pulsating protrusion of the

eyeball, with great œdema of the lids and conjunctiva—a condition rarely if ever met with except after cavernous sinus thrombosis—made it seem most probable that a basal fracture had occurred with resultant laceration of this vessel, which had not thrombosed and produced the proptosis until the third day.

It seemed to us both that a craniotomy offered the only hope of recovery. The child was transferred to Dr. Williams' department

FIG. 3

CASE III.—View of cranial vault, to show scars of the bilateral operation.

at the hospital, where an operation was performed early on the following day, October 12, 1904.

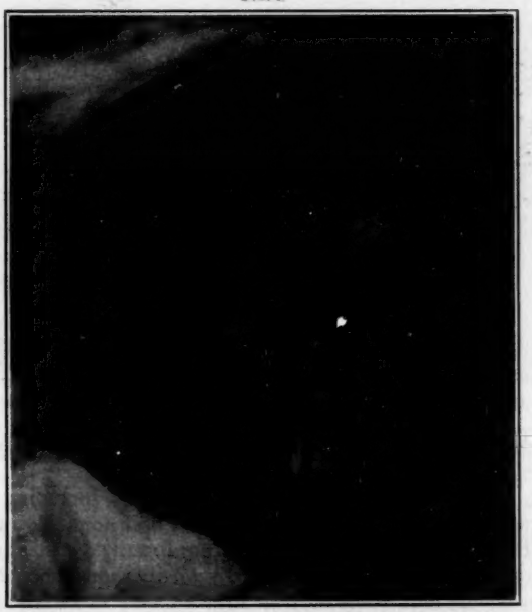
As in the previous cases, an osteoplastic flap, including most of the parietal bone, was elevated. This was first done upon the right side. The dura was found to be extraordinarily tense, and, in the endeavor to reflect this membrane by an incision concentric with the osteoplastic flap, some cortical injuries were unavoidably made, owing to the great cerebral tension, which caused the brain imme-

diately to bulge through the small primary opening. This protrusion was so extreme, and the soft, jelly-like infant's brain so easily lacer-

FIG. 4

CASE III.—Lateral view, to show size and position of one of the symmetrically placed osteoplastic flaps.

FIG. 5



CASE III.—Photograph, taken during sleep, of patient two months after operation, to show complete retrocession of exophthalmos. Contrast with Fig. 2.

ated, that it was impossible to elevate the temporal lobe sufficiently to discover whether or not a clot was present at the base as a result of the presumed lesion in the cavernous sinus. The operator had to be satisfied with nothing more than washing off from the cortex of the numerous old blood clots which were present on the exposed surface, as well as those which could be liberated from below by such slight dislocations of the hemisphere as was possible without occasioning further cortical lacerations. With the removal of these clots and the escape of cerebrospinal fluid, the bulging, though still considerable, diminished to such an extent that the dura could be brought together again by sutures. Being fearful, however, lest the tension was sufficient to break down these sutures and lead to a hernia cerebri, or, worse, to a fungus, the desperate nature of the case seemed to justify a similar exposure of the other hemisphere. Consequently, a parietal flap was turned down in like fashion on the left side, and a similar procedure carried out, with the washing away from the surface of numerous blood clots and the escape of cerebrospinal fluid. This sufficed to diminish the tension to such an extent that the fontanelle became depressed, and the brain no longer tended to protrude through the dural openings. These were carefully approximated by sutures, the bone-flaps were replaced, and the scalp wounds were closed without drainage.

The child, contrary to our expectations, and almost against our desires, immediately began to improve. By the following day nourishment was well taken from a bottle and she soon began progressively to put on weight. Both wounds healed by primary union (Figs. 3 and 4). It became evident after a few weeks that the cavernous sinus was once more becoming re-channelled. Ultimately, the circulation from the eyeball became re-established, and the exophthalmos gradually disappeared (Fig. 5). It has, however, probably left the eye completely blind.

It is, of course, difficult to tell, before a few years have elapsed, what will be the mental status of the unfortunate child whose life seems to have been saved in this way. At the present time, however, it seems to be developing normally, and is a natural and healthy-appearing baby, with no evidences of motor disability.

Case IV.—The patient, a second child, was born on the evening of Tuesday, February 14, 1905, after a brief and rather precipitous labor. It was first seen three days later in consultation with Dr. Iglehart, who, though not present at the labor, saw the child soon after birth, and at that time believed it normal in every respect. On the following day, however, his attention was called by the nurse to a swelling which had appeared over the left parietal region. This was somewhat tense and fluctuant, and presented the appearance of a cephalhæmatoma. This swelling during the day, February 15th, and on the following day, increased rapidly in size, until by

the evening of the third day it had become as large as a hen's egg. Beyond this, nothing was thought to be wrong with the child until Friday, February 17th. On this day it was first noticed that the left pupil was larger than the right, and that the pulse had become slow. The nurse also noted that the child had become stupid in taking its nourishment. Fearing that the symptoms indicated an intracranial hemorrhage, Dr. Iglehart made every effort to secure an early consultation; there was an unfortunate but unavoidable delay of some hours, and during the interval the child had become very much worse.

When first seen the large, tense swelling on the left side of the head, its base corresponding exactly in size, position, and shape to the left parietal bone, gave the child a very remarkable appearance. The swelling projected from the surface of the head fully 5 cm. It was pulsating. Pressure upon it did not seem to affect the infant in any way. The left pupil was dilated and the left eyelids kept closed. The eyeballs were somewhat prominent, though there could be said to be no definite exophthalmos. The eye-grounds were not examined. The superficial veins radiating from the angles of the eyes, and particularly those running over the scalp, were so much dilated as to be visible at a distance. There were numerous small, punctate hemorrhages in the skin over the neck and chest. The child's right side was possibly more rigid than the left, but this could not be positively stated. There had been one severe general convulsion about four in the afternoon. The child had taken practically no nourishment for twelve hours.

As rapidly as possible preparations were made for the operation. While the head was being shaved the infant had a second general convulsive seizure, accompanied by several sharp cries. Its general condition was very unfavorable.

Operation, February 17, 1905, at 10 P.M. The head was carefully shaved and prepared, every effort being made meanwhile to sustain the infant's bodily warmth and strength. Chloroform was used as the anæsthetic, a few drops only being required. A needle was first inserted into the cephalhæmatoma and dark fluid blood flowed out of it. An incision was then made about its base, concentric with the left parietal bone. An amount of clotted and dark fluid blood which must have measured two or three ounces was evacuated. This clot lay between the epicranium and the denuded shell of the parietal bone. There was no indication of any connection between it and the intracranial contents. After the evacuation of this clot and exposure of the parietal bone, the latter was turned back almost in its entirety as in the preceding cases. As was expected, a tense, plum-colored dura, exactly similar to that seen in Case I., was brought into view. The dura was opened and a large clot about a centimetre in thickness, which seemed to cover the entire hemisphere, was disclosed. Much of this clot escaped as soon as the opening was

made, and other large pieces of it were lifted off from the surface with a blunt instrument and removed.

Possibly I should have been satisfied with the relief which this partial operation would have afforded. It doubtless would have been wise to have postponed further interference to another and later occasion, but the child's condition seemed to justify an effort to more thoroughly remove the clotted blood which was within easy access over the anterior and posterior poles of the hemisphere. While irrigating away some of the blood, the child suddenly stopped breathing. Under artificial respiration the heart beat continued for some minutes, but all efforts to restore spontaneous breathing were unavailing,

Postmortem Examination, February 18th. A careful dissection made it quite evident that the cephalhæmatoma had no direct connection whatsoever with the subdural hemorrhage. It seems quite likely that its formation was due to the fact that during the precipitous labor there was a slight injury and slipping of the epicranium from the parietal bone; this did not lead to an extracranial hæmatoma until the intracranial hemorrhage reached a size sufficient to produce great venous engorgement, and this in turn caused bleeding from the torn venules of the epicranium, which under other circumstances would not have had an intravascular tension sufficient to lead to so extensive an extravasation. The outlines of this extracranial hemorrhage were found to be limited by the attachment of the epicranium at the margin of the parietal bone. There was no extracranial extravasation over the frontal or occipital bones. On removing the calvarium it was found that the hemorrhage was limited to the left side of the cranial cavity, and was almost entirely supratentorial. The cerebrospinal fluid over the right hemisphere and in the spinal canal was somewhat bloody, but there were no clots. The accompanying photograph (Fig. 6), taken with the brain still *in situ* but after removal of the calvarium, shows the widespread distribution of the clot. At one place only, the convolutions are exposed; namely, where the clot had been partially removed at the time of the operation. On clearing away the remainder of the clotted blood, which surrounded the hemisphere like a mould, thickest in the central regions, the cortex was found to be macroscopically uninjured. One of the larger cerebral veins was found torn away from the dura at its point of entry into this membrane near the longitudinal sinus.

It seems needless to add anything to the story which these cases tell. There are, however, certain points which may deserve mention before closing this paper. One of them is that a new-born child, with proper regard for hæmostasis and careful avoidance of exposure, will stand a cranial operation well. This is contrary, however, to the statements in our text-books; but when one considers what an amount of traumatism every child must have to endure while making

its entrance into the world, it would seem reasonable to expect that a careful operation, provided there is no loss of blood, would be no more serious. An operation could hardly have been undertaken under less favorable conditions than those presented by the third one of these patients. Yet the child was in better condition a few hours after the double craniotomy than before the operation. Further experience alone will determine the best method of treating the patients with extensive hemorrhages such as the first and last of these cases presented. The operation in each instance should have

FIG. 6

CASE IV.—Postmortem appearance of brain after removal of calvarium. Note the extravasation over the entire left hemisphere, the convolutions of which are visible in the parietal region alone, where a portion of the clot was removed at the operation. Right hemisphere totally free from extravasation.

been done at least a day earlier, and, as the results show, I should have refrained from attempting too much at one sitting.

An argument, advanced against the possibility of carrying out these operations on the new-born, is that the blood of these infants has such a slow coagulation time that a fatal hemorrhage from the incision would result. This is no more true for cranial operations in the new-born than for those in other parts of the body, and my experience leads me to think that coagulation is as rapid as in the adult, though I have made but few accurately timed tests. It is

worthy of remark in this connection that the effused blood in all of these cases has been found clotted.

It will have been noted that in two of the patients symptoms definitely pointing to intracranial trouble did not appear or were not recognized until several days after birth. A more careful study of symptomatology—and this will surely follow upon the knowledge that something immediate can be done to relieve these cases and possibly to ward off the consequences of the hemorrhage—will perhaps enable us to recognize these conditions earlier.

In three of the cases—those with primary cortical hemorrhages—the effusion was confined to one side. The birth palsies, however, are more commonly supposed to be bilateral, and many of the writers on the subject regard the spastic diplegias or paraplegias as the typical cases of Little's disease, whereas a spastic hemiplegia is supposed to be more commonly of infectious origin. Extensive as it was, however, the effusion in Cases I. and IV. was unilateral, and had these patients survived, unoperated, a spastic hemiplegia probably would have resulted. In Case II., on the other hand, symptoms, appearing on the fifth day after birth with fever and unilateral convulsions, might easily have been ascribed to some infectious process; yet a hemorrhage was found and they immediately abated after the operation.

If it can be demonstrated that a craniotomy on the new-born child, when conducted with due precaution and delicacy of manipulation, is comparatively free from danger—and Cases II. and III. show how well such operations may be tolerated—I believe the immediate risk of death and the sorry late consequences of meningeal birth hemorrhages may be avoided in many cases by surgical interference. It is reasonable also to suppose that these explorations will at the same time lead us to a better understanding of the varying pathological features of this group of diseases in their early stages.

INTESTINAL PERFORATION IN TYPHOID FEVER IN EARLY LIFE.[1]

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The occurrence of typhoidal perforation in children deserves consideration, first, on account of its supposed rarity, and, second, on account of the difficulty of recognition which is often presented and which seems to me to be largely peculiar to this time of life.

As to the *rarity* of its occurrence there appears to be little variation of opinion among many of the leading text-books. That the accident

[1] Read before the Association of American Physicians, May, 1905.

is uncommon, although just to how great an extent, is a matter regarding which statistics vary. Marfan[1] says that perforation in children is almost never observed. Baginski[2] speaks of it as uncommon, and has seen but 2 cases. Rotch[3] considers it exceedingly rare, and Holt[4] has found but 12 instances in 1028 reported cases of typhoid fever in children. As 4 are said to have recovered we are uncertain of the diagnosis in these. Heubner[5] has never seen a case. Wilson[6] considers it exceedingly rare, and Koplik[7] and Gerhardt-Seiffert[8] are of the same opinion. Henoch[9] has seen it but once in all his cases. Curschmann[10] speaks of perforation as rare in children.

On the other hand Ashby and Wright[11] have met 4 cases, and Méry,[12] although admitting its great rarity, thinks that this has been somewhat exaggerated. He says that at least 14 or 15 cases have been reported from the Children's Hospitals of Paris during the last six years, quotes 26 perforations in 1506 cases collected by Setbon, and says that Murchison, in 73 reported perforations, found 10 in children under ten years of age and 8 in those from ten to fourteen years.

Journal literature, too, although in general speaking of the rarity of perforation in early life gives somewhat varying statistics. Roemheld[13] found 3 perforations in 117 cases, and Montmollin[14] reports 7 cases, 3 of which, however, recovered. Morse[15] reports no perforations in 234 cases, and only 4 in 735 cases published by eight observers whom he quotes. Fitz[16] found 7 perforations among 192 children, and Adams[17] 11 perforations in 337 cases. Rennert[18] gives 4 perforations out of 471 cases, and Schulz[19] 8 cases of perforation in children.

One of the most valuable contributions to the subject is that of Elsberg.[20] Out of 289 collected cases of operation for perforation in typhoid fever, 25 occurred in children less than fifteen years of age. He gives a useful analysis of the symptoms exhibited in these and concludes that they do not differ materially from those occurring in adult life. This article deals so thoroughly with the

[1] Traité des maladies de l'enfance (Grancher, etc.), 1897, vol. i. p. 321).
[2] Lehrb. der Kinderkr., 7 Auf., pp. 201, 211.
[3] Pediatrics, 1901, p. 451.
[4] Diseases of Infancy and Childhood, second edition, p. 1054.
[5] Kinderheilk., vol. i. p. 508.
[6] Cyclopedia of the Diseases of Children. Keating, vol. i. p. 408.
[7] Diseases of Infancy and Childhood, p. 173.
[8] Lehrbuch der Kinderkr., p. 175.
[9] Kinderkrankheiten, 8 Auf., p. 780.
[10] Nothnagel's Encyclopedia, p. 333.
[11] Diseases of Children, second American edition, p. 283.
[12] Traité des maladies de l'enfance (Grancher, etc.), second edition, vol. i. p. 495.
[13] Jahrb. f. Kinderheilk., vol. xlviii. p. 207.
[14] Thèse, Neuchatel, 1885.
[15] Boston Medical and Surgical Journal, Feb. 27, 1896.
[16] Transactions of the Association of American Physicians, 1891.
[17] Archives of Pediatrics. Feb., 1904.
[18] Deutsch. med. Wochen., 1889, p. 1063.
[19] Jahrb. der Hamburger Stadtskraukenanstalten, 1889, vol. i.
[20] Annals of Surgery, July, 1903.

literature of the subject that it is not necessary to enter more fully upon it in this connection.

That typhoid perforation should occur less frequently than in adults is in accord with the characteristics of the lesions of the disease in early life. In the fœtus typhoid ulceration is uniformly absent. In infants it is exceptional that it is at all extensive or severe. As age advances it grows more common, but is generally less frequent and less severe than in adult life. Nevertheless, very extensive ulceration can exceptionally occur even in early childhood, as I have seen proven upon the post-mortem table. Consequently, the occasional development of perforation is to be expected as a matter of course, and the older the child the more liable is this to happen.

As my experience has been unfortunate in having met with 6 instances of perforation in children, it may be of service to put these on record. The history of the first has been previously published, but is reported here for the sake of completeness of the group. The others I have not recorded heretofore, although the fact of their existence I had referred to in the discussion of a paper by Dr. J. H. Jopson.[1] The surgical aspects of the sixth case have been alluded to briefly by Martin and Pennington.[2] For various reasons none of the cases had a leukocyte count made when perforation was suspected. It is doubtful, however, whether this, if done, would have aided much in the diagnosis.

Case I.—Leila B., aged ten years, entered the Children's Hospital of Philadelphia, February 26, 1897, on about the sixth day of typhoid fever. During her first two weeks in the hospital she passed through an ordinary attack of the disease without abdominal pain or tenderness and without diarrhœa until March 12th, when two watery movements occurred. On the evening of March 15th, approximately the twenty-third day of the disease, rather severe pain developed in the right iliac region, yet without signs of any serious trouble being present. The temperature had been approximately 104° unless reduced by tubbing. During the night slight intestinal hemorrhage occurred, with a fall of temperature to 103°. On the following day, March 16th, there was abdominal pain, not very severe, with slight distention of the abdomen, accompanied by tenderness and increased resistance. The pulse had lost in force, and the face had a somewhat anxious expression; yet there was no evidence whatever of collapse, the temperature remaining about the same. Later in the day vomiting began and continued troublesome. The abdomen was more distended and the hepatic dulness was nearly obliterated. The temperature had fallen in the evening to 101°. During the following night the abdomen became more distended and the child died in the early morning of March 17th, the temperature having fallen slowly to

[1] Archives of Pediatrics, March, 1904.

[2] American Medicine, Nov. 21, 1903.

99.6°. None of the ordinary symptoms of collapse had been exhibited at any time. The occurrence of a perforation had been suspected at the beginning, and later believed to be undoubted, but operation could not be performed—for reasons upon which it is not necessary to enter.

The autopsy showed a perforation of the ileum, about fifteen inches above the ileocæcal valve, with extensive and deep ulceration of Peyer's patches.

CASE II.—Charles K., aged eleven years, entered the Children's Hospital on February 28, 1899. He had been ill for four days with fever, malaise, chilly sensations, loose cough, and some pain in the abdomen and hips, "a kind of attack that he had frequently had before." An examination on March 3d showed a slight diminution of expansion of the left side of the chest, with dulness on percussion from the sixth rib downward in front and in the axilla; somewhat feeble respiration and some friction sounds on deep inspiration. Posteriorly there was dulness on percussion over the lower third of the left lung. The spleen could not be felt. The diagnosis of pleurisy or of pleuropneumonia was made.

The boy continued to do well, the temperature remaining elevated from 100° to 103°, and the bowels, at first normal, becoming diarrhœal in character. On the afternoon of March 6th, his seventh day in the hospital, and apparently his eleventh day of illness, he complained of pain in the abdomen and vomited. The symptoms were apparently the result of his having eaten some ice-cream. Vomiting continued all night, and general weakness developed, with feeble pulse, sufficient to make hypodermic stimulation necessary. The temperature arose in the mean time to 105.8° without any previous fall of it. March 7th found him with constant vomiting, a cold and clammy skin, and general abdominal distention, tenderness, and pain. The friction sounds over the lung were less numerous. The condition continued all day, the cause of it being not at all understood. His temperature ranged from 102° to 105°. On March 8th he was better, but the features were quite sunken. The abdomen was less distended, but very tender, and the hepatic dulness not at all diminished. The dulness over the base of the left lung had disappeared. A few small rose spots were discovered on the trunk, suspiciously like the typhoid roseola. There appeared to be some increase of resistance in the right iliac region. The temperature was 106° in the morning of this day and 100.6° in the evening. The existence of typhoid fever was now suspected for the first time, and it seemed very certain that perforation had occurred. No operation could be performed.

At 3 A.M. on the morning of March 9th the temperature dropped to 97.8°, but quickly rose, and at 7 o'clock A.M. it was 101.2°. Failure of strength continued; on March 10th there were sunken features, jactitation, a tense and moderately distended abdomen,

and a greater resistance to palpation of the right iliac fossa. The temperature in the morning was 105.4°, but fell to 102.4° in the evening, when death occurred.

Autopsy showed a general purulent peritonitis; a perforation in the ileum five inches above the ileocæcal valve; other ulcers in the ileum, some apparently in process of healing; evidences of quite recent pleurisy over the right lung, and some soft adhesions over the base of the left lung.

It was evident from the autopsy that the disease was farther advanced than the history indicated, and that the child had been a case of walking typhoid, complicated later by pleurisy.

Case III.—Celia S., aged twelve years, first seen in consultation with Dr. S. N. Dubin, May 1, 1899. The child was at the time apparently in the fourth week of typhoid fever. Her mind had been clear and her condition good throughout, except for constant tendency to hyperpyrexia still existing, the temperature being often 106° or over. The tongue was moist and there were slight tremulousness and moderate diarrhœa. By May 4th her condition was much the same, except that the diarrhœa had nearly ceased. There was, however, retching when food was given. Later, vomiting began again, but was only occasional, and the child's condition continued no worse. There were at times specks of blood in the stools. At five o'clock on the morning of May 9th she suffered from sudden severe abdominal pains, and a fall of temperature from nearly 107° to 97°. Dr. Dubin felt certain that she had a perforation. I was asked to see her again, and did so in the afternoon, but failed to meet Dr. Dubin, who was unavoidably detained. She had at this time moderate abdominal tenderness on pressure, but no distention or vomiting, and no evidence whatever of collapse or any noteworthy abdominal pain. Her temperature was still elevated. I had heard nothing of her suspicious symptoms, and careful questioning of the parents, who were rather ignorant people, failed to elicit any history of their previous presence. The indications of perforation returned later, and she was removed, on May 10th, to the Methodist Hospital, where I saw her again in consultation with Dr. Dubin and Dr. E. W. Holmes, the attending surgeon. She was now clearly very ill, and her mind was partly clouded. Operation was done at 10 P.M., about forty-one hours after the first symptoms appeared. A perforation was found in the ileum, with several ulcers threatening rupture. Death occurred before morning.

Case IV.—Frank M., aged eight years, was admitted to the Children's Hospital of Philadelphia January 2, 1903, apparently at the end of the first week of typhoid fever, having suffered from loss of appetite, malaise, slight cough, headache, and slight abdominal pain. Examination showed the child emaciated to some extent, the heart normal, a few rales in the lungs, the spleen

indistinctly felt, rose spots present on the abdomen, and slight gaseous distention. His condition continued fairly good, there being very slight diarrhœa, and the temperature running from 102° to 104° or 105° and making hydrotherapy necessary. On January 14th, the thirteenth day of his stay in the hospital, he was still doing well, although having a rather severe attack of the disease, but at 11.30 P.M. vomiting occurred, the temperature being 104°, and continuing from this to 105° during the night. On the morning of the 15th his pulse was weak and irregular and his abdomen moderately distended and only slightly painful on pressure, but extremely tense. The hepatic dulness was normal. There seemed to have been no sudden development of this condition, and his temperature did not fall below 101.2° on this date. He vomited again at 9.45 A.M. and at 11.30 A.M. The possibility of a perforation having occurred was considered, but the absence of any evidence of collapse and the slight degree of vomiting and abdominal tenderness and distention, combined with the presence of normal hepatic dulness, made it seem doubtful. There was, however, a suspiciously anxious expression of face. Request was left that Dr. R. Le Conte, the surgeon on duty, examine him when he made his visit, and a message was sent to his parents asking permission to operate if necessary. The child's symptoms grew worse so rapidly that a few hours made the diagnosis practically certain. The distention of the abdomen increased greatly, as did rigidity and tenderness, the liver dulness disappeared, abdominal pain became extreme and continuous, vomiting returned and occurred repeatedly, and the features grew very much sunken. The temperature continued at about 103°, but diminished to 100.4° in the afternoon. Operative interference was absolutely refused by the parents, who removed the child on the following day, January 16th, against advice. He died on January 17th.

Although no autopsy was held, there can be no reasonable question of the diagnosis.

CASE V.—Mary B., aged four years, was admitted to the Children's Hospital of Philadelphia March 8, 1903. Ten days previously she had commenced to suffer from loss of appetite, malaise, headache, diarrhœa, and epigastric tenderness or pain. After a short time the parents noticed that she had fever, and she was then put to bed. During the last three days the temperature had reached 104° and the other symptoms had continued. On admission she exhibited some distention of the abdomen, enlargement of the spleen, roseola, and a coated tongue. The mind was clear; the Widal reaction was positive. She was considered to be probably in the middle of the second week of the disease. The attack ran rather a mild course, the abdomen showing considerable tympanitic distention, the child looking bright, and the temperature gradually falling. By March 17th, the last occasion on which I

saw her, the patient was convalescent, the temperature having been nearly normal most of the time for a week. I give a synopsis of the ward notes made after this date: At 7 A.M. on the morning of March 18th the child vomited food and medicine and had slight abdominal pain, but her temperature remained at 99° and she did not look ill. At 9.15 A.M. she appeared feverish and vomited again. At noon her temperature was 100°. There was no abdominal pain, distention, or tenderness, and the child was sitting up in bed in apparently good condition, At 3 P.M. the temperature was about 103° and there were still no abdominal symptoms.

Shortly after this she vomited again twice, at 3.15 and at 4 P.M. At 6 P.M. the temperature was 105.6°. The mind was clear, but the child looked ill and was somewhat restless. There was no abdominal distention or pain. By 9 P.M. the temperature had risen to 106.6°, the child was growing rapidly worse, and died at six o'clock on the morning of March 19th. Distention, pain, and tenderness of the abdomen had continued to be absent, and there had been no return of vomiting since four o'clock on the preceding afternoon.

The autopsy showed a purulent peritonitis. The ileum exhibited numerous small ulcers near the ileocæcal valve, and in one of these was a minute perforation. The serous layer of the intestine at this point was adherent to another knuckle of the gut, thus partially closing the perforation.

CASE VI.—Esther S., aged six years, seen in consultation with Dr. Joseph Neff April 4, 1903. The child was presumably in the second week of typhoid fever. Rose spots were present. The general condition had been good. About four o'clock in the afternoon of this day she had suffered from quite severe abdominal pain followed by vomiting, and was said to have had decided depression of strength, although without actual collapse. It is not known whether there was any fall of temperature. I saw her at 10 P.M. and found her in excellent general condition, with the pulse of good quality, no anxious expression of face, slight diarrhœa, no abdominal pain, tenderness only on firm pressure, and distention only slight. Vomiting had ceased. The diagnosis of a perforation was considered, but dismissed as too uncertain to warrant operation.

I therefore advised cautious delay, but immediate operation should any of the symptoms return. A telephone message at 9 A.M. on the next day, April 5th, told me that the child had had a good night, that there had been no return of pain or vomiting, and that the abdomen was not distended or tender. Later in the morning, however, at about eleven o'clock, abdominal pain returned, distention became decided, and the child was removed to the surgical ward of the hospital of the University of Pennsylvania. Here the symptoms continued, perforation was suspected, and operation was done by Dr. Edward Martin at about 5 P.M. in

the afternoon of the same day. One perforation was found and two ulcers threatening rupture. The child was in bad condition immediately after the operation, but by the next day all symptoms were favorable. She did well for a time, but evidences of the formation of a pus cavity developed, and, although this was found and evacuated, her strength failed and death occurred seven days after the first operation.

I have referred in opening the subject to the difficulty in the recognition of perforation frequently exhibited in early life. Some of the cases here reported possessed the classical symptoms as usually seen in adults; others certainly illustrated the difficulty to which reference has been made. Case I., although exhibiting abdominal pain, tenderness, and distention, had at first no more of these than is common in cases of typhoid fever with tympanites. There was no initial fall of temperature or evidence of collapse. The symptoms as exhibited could be called suspicious only. Later in the same day I should certainly have advised operation had it been possible to have had it performed, as there was reason to believe that perforation was probably present.

Case II. was absolutely misleading throughout. Even the presence of typhoid fever was not suspected. The child was supposed to be suffering from pleurisy only. The occurrence of vomiting and abdominal pain seemed clearly connected with an indiscretion in diet. It was only on the day following that on which perforation probably occurred that the suspicion arose that something more was ailing him, and not until the succeeding day that the diagnosis of typhoid fever and perforative peritonitis was entertained. The symptoms by this time were sufficiently positive to make operation urgent, although even now the abdominal distention was not great, the only fall of temperature below normal having occurred after the perforation had existed, probably, over two days.

A most unfortunate circumstance prevented a better understanding of the symptoms in Case III. Those at the onset were clearly those of perforations, or possibly of concealed hemorrhage. The failure of blood to appear later in any large quantity in the stools excluded the latter. Within a few hours all symptoms had disappeared to such an extent that, although entertaining the thought of the presence of perforation, based upon the existence of some abdominal tenderness, I did not feel warranted in believing that this had occurred. Had I known of the earlier condition I should, of course, have advised immediate operation.

Case IV. presented doubt only during the first hours of the condition. There was at that time no history of collapse and no more abdominal distention, pain, and tenderness than is very frequently present in cases of typhoid fever. There was also no loss of hepatic dulness. The earliest really suspicious symptom was alteration

of the facies. In a few hours the symptoms were beyond question.

It is difficult to say in Case V. on what a diagnosis of perforation could possibly have been based. The vomiting was only occasional; the complaint of pain had been temporary and not great. There was no collapse, and throughout the twenty-three hours which followed until death occurred there was neither abdominal tenderness, distention, nor pain to guide us. The count of the leukocytes might have been of significance if there had been any suspicion of what ailed the child, but even this is by no means certain.

Case VI. presented elements of great uncertainty. In view of the fact that vomiting and abdominal tenderness and pain are of such frequent occurrence in typhoid fever, I could not feel warranted in making a diagnosis of perforation or in advising exploratory laparotomy, especially when the symptoms had been unattended by any certain evidence of collapse and had so largely disappeared at the time of my visit. In any similar case I should, I think, again follow exactly the course taken in this—viz., delay cautiously until a diagnosis of perforation was more probable. From the point of view of the immediate results of the operation the child did well. Death occurred rather from a sequel.

A CASE OF ACUTE LEUKÆMIA, WITH SOME STRIKING CLINICAL FEATURES. OBSERVATIONS ON METABOLISM IN THIS CASE, AND IN A CASE OF SEVERE PURPURA HEMORRHAGICA.[1]

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My chief purpose in this report is to discuss certain disturbances of metabolism in leukæmia and purpura hemorrhagica that are of much clinical and pathological importance, and to inquire somewhat into the nature of these disturbances, particularly their relation to the hemorrhage that occurs in both these diseases. I shall, however, first mention briefly some striking clinical features in the case of acute leukæmia, which are of interest from an etiological, and particularly from a diagnostic, standpoint.

There was an apparent connection of the beginning of the symp-

[1] Read at the meeting of the Association of American Physicians, Washington, D. C., May 16 and 17, 1905.

toms with the extraction of a tooth, and therefore with the opportunity for infection, a point that is of some interest in relation to the etiology of the condition; and the clinical resemblance of the case to diphtheria and angina Ludovici has serious diagnostic importance. In this instance the possibility that the disease had originated from mouth infection set up through the extraction of a tooth must, however, be dismissed as extremely improbable. The very early appearance of severe stomatitis in many cases of acute leukæmia has led to the repeated suggestion that the disease itself, or at least the rapidity of its progress, is caused by infection, particularly through the diseased mouth; and in a number of instances, as in my case, the extraction of a tooth was apparently directly followed by rapidly progressing acute leukæmia. In the instance that I am describing, however, the actual conditions were almost certainly the reverse of this; that is, the disease itself, in its incipient stage, evidently produced the symptoms that led to the extraction of the tooth.

As to the appearances suggesting diphtheria and angina Ludovici, there is, I think, in systematic writings dealing with acute leukæmia, or in more fragmentary papers, little, if any, discussion of the possibility of mistaking this disease for diphtheria, and there is no sufficient consideration of the possibility of mistaking the disease for simple angina Ludovici without other general disorder. Late in the course of this case it would have been wholly impossible without bacteriological investigation to have stated that it was not diphtheria; and, on the other hand, angina Ludovici seemed, when I first saw the man, to be probably the main condition present until the blood examination, that was made immediately afterward, showed at once that he had acute leukæmia. In the classes in pathological anatomy in the General Hospital at Vienna, specimens from several cases of acute leukæmia have, I am told, been exhibited in recent years with the statement that they had been mistaken for some time for angina Ludovici. This mistake is a natural one, and could scarcely be avoided in some instances until the blood condition became distinctive. Indeed, there is every opportunity for patients with acute leukæmia to develop actual Ludwig's angina, the condition of the mouth that is so common in this disease giving ready opportunity for infection of the tissues of the neck.

Without giving extensive details of the clinical side of the case of acute leukæmia, I would refer to the following points: The patient, a man aged thirty-six years, was seen with Dr. Cryer on January 15, 1901. He gave a good family history, and a personal history that was of no interest until about three weeks before I saw him. For some months previous to this time he had been under severe business and emotional strain, but no results of any consequence had been noted. Late in December, 1900, the first symptoms of importance appeared. He then had swelling of the gum

about the upper posterior molar tooth on the right side, together with much pain, and his dentist extracted the tooth. The fact of interest at this point is that the trouble with the tooth was almost certainly due to leukæmic infiltration of the tissues about it. The history first obtained from the patient apparently indicated the contrary—that the disease in the mouth and elsewhere had followed the extraction of the tooth, for subsequent to its extraction he had local ulceration of the gum, of unhealthy appearance and with necrotic pseudomembrane over its surface, and this ulceration advanced until it involved a large part of the surface of the gums of the left side of the mouth. At this period, too, the man noticed the first disturbance in his general health. The tooth, however, was healthy, and there was at that time no disease of the surrounding tissues, the necrosis having followed the extraction merely because of the disease that had already begun. It seems probable that had this point been ascertainable, it could have been shown in at least a considerable portion of the other similar cases in the literature that the initial symptoms of leukæmic infiltration actually preceded the development of the local opportunities for infection.

Because of the dental condition the patient was seen in consultation by Dr. Cryer a week after the tooth was extracted, and ten days later, after a telephonic communication, Dr. Cryer had him admitted to the private ward of the University Hospital for examination, thinking that the condition of the mouth might be due to an impacted tooth. It was, however, at once apparent that he had serious general disease, and at Dr. Cryer's request I took charge of him. In the period intervening between this time and his previous visit to Dr. Cryer he stated that there had been marked and rapid loss of strength, with a good deal of emaciation and mental depression; and he had also had decided disturbance of vision and hearing, having had difficulty in reading even the large print of a newspaper, and at times in recognizing the members of his family, and having become so deaf as to be uncertain about ordinary conversation. For some days before entering the hospital he had had much pain on attempting to masticate or swallow, and had had a slight, thin, bloody discharge from the nose; he had also become extremely hoarse, and had had difficulty in inspiration. There had been no other noteworthy symptoms. At the first glance the immediate suggestion was that he had a severe diphtherial infection. He was evidently much prostrated; the pulse was rapid and weak; he had marked inspiratory stridor, and his voice was a hoarse whisper; there was a thin, blood-tinged discharge from the nose; a little pseudomembrane was at once visible in the nose, and he evidently had distress upon swallowing. His temperature was 101.2°. General examination at once made the possibility of diphtheria very questionable; it showed the following conditions: He was weak and seemed emaciated; there were small ulcers, with

hemorrhagic bases on the lips and about the nostrils; there was a narrow line of ulceration of the mucous membrane of the upper jaw, most marked posteriorly, but extending to the incisors; there were similar patches of ulceration on the lower jaw, though less extensive, and there were small ulcers beneath the tongue and on the dorsum of the tongue. All these ulcers had hemorrhagic bases, but were not bleeding. Their surfaces were covered with a dirty looking, yellowish-gray pseudomembrane-like necrotic deposit. The gums were swollen and congested, but did not bleed upon manipulation; the tongue was somewhat swollen. The pillars of the pharynx and the walls of the pharynx itself were very much swollen, and the tonsils were so large that there was a space of only half an inch between them. The lumen of the pharynx also seemed very small, as a result of the swelling of the tissues. There was, however, no redness, pseudomembrane formation, or ulceration back of the tongue. The nose was nearly blocked on both sides, the mucous membrane swollen and somewhat congested, and there were small ulcerated areas which showed a pseudomembranous deposit.

The man's neck appeared to be much swollen, particularly on the left side, and the tissues, on the left side especially, felt dense and infiltrated half-way down to the clavicle. Part of this was evidently due to glandular enlargement, one gland, about one and one-half inches in length, being readily felt, as well as a number that were smaller; but the swelling of the neck was so conspicuous as to be evident at the first glance, and it appeared to be due to a considerable extent to diffuse infiltration of the tissues, and not simply to glandular enlargement.

The glands elsewhere could be everywhere felt, and in the axilla several were about an inch in diameter; the spleen could not be palpated, but the dulness was enlarged. Examination elsewhere showed nothing of diagnostic importance. The lungs were decidedly congested at the bases; the heart was a little dilated, the sounds weak, the action rapid. The man had difficulty in reading even the head lines of a newspaper, and he could hear a watch at but six inches distance with the left ear; three inches with the right.

A culture was taken at once, and proved negative for diphtheria bacilli. The resident physician's blood count, which was immediately confirmed by Dr. C. Y. White, showed 466,640 leukocytes; 1,820,000 red cells. The hæmoglobin estimations at this time and subsequently were unreliable because of the immediate clouding that took place.

The examination of stained specimens showed a very definite but somewhat peculiar picture of acute lymphatic leukæmia. I shall not discuss the morphology of the blood, as I have asked Dr. White to include the study of this blood in discussing a series of cases that he is collecting.

The conditions that I have mentioned show that there was a

strong suggestion of diphtheria and also of angina Ludovici in the case. Twenty-four hours after I first saw him diphtheria could certainly not have been excluded without negative cultures; even the definite determination that acute leukæmia was present would scarcely have finally excluded diphtheria, for at that time he had pseudomembrane on both tonsils, and he had the nasal and laryngeal symptoms that are so common in diphtheria.

The further progress of the case was one of increasing prostration, with death two days and a half after I first saw him, from cardiac failure, œdema of the lungs, and sudden collapse. The course was interesting in only two particulars: In the first place, the obstruction of his larynx grew more and more severe, and finally became so marked that the possible necessity for tracheotomy was kept anxiously in view, though this measure would have been most reluctantly undertaken because of the hemorrhagic tendency. So far as I know, marked laryngeal obstruction has not been described in acute leukæmia, though it has repeatedly been seen in chronic lymphatic leukæmia. The laryngeal symptoms were, as was expected, found to be due to extensive infiltration of the larynx, with marked swelling and slight ulceration of both the false and the true vocal cords.

The other point of interest was the hemorrhage. The man exhibited no hemorrhagic tendency, excepting as a result of the second puncture made for study of his blood. This minute wound was followed by oozing of blood in almost a continuous stream for the greater part of an hour; all measures used to stop it were ineffectual, and the amount of blood lost was certainly as much as a pint. This feature is interesting chiefly in relation to the manner in which the hemorrhage was produced. Grave hemorrhage from the same cause has previously been described in rare instances.

A few hours after his admission, when the diagnosis of acute leukæmia had been made, a metabolism experiment was begun. This, unfortunately, could be continued for but one period of twenty-four hours, because it became impossible after this to save his urine, owing to his profound prostration and mental obscurity. In this time, however, sufficiently characteristic conditions were observed.

He took during this period only milk and broth. The nitrogen content and the amount of P_2O_5 in these were determined. I also determined the nitrogen and P_2O_5 in the urine: estimations of the uric acid and purin bases were begun, but, unfortunately, were lost through the mistake of a laboratory servant.

The man's food contained 7.25 gm. of nitrogen, 1.84 gm. of P_2O_5. The urine during the same period contained 29.534 gm. of nitrogen and 3.056 gm. of P_2O_5. While I lost the estimations of uric acid and purin bases, I think I am safe in saying, from the large precipitate produced by ammoniacal silver nitrate, that the uric acid was greatly increased; it was probably above 2 gm., at any rate.

The feces could not be accurately collected, and hence the nitrogen and phosphates in the feces were not determined; there was probably more than a gram of nitrogen in the feces, however. In addition to this, the man lost during this period, as I have stated, a large amount of blood, and this must have contained 4 gm. or more of nitrogen. The balance between the intake of nitrogen and the outgo in the urine alone shows a loss of 22.28 gm., and with the loss from other sources he must in all have had a negative nitrogen balance during this twenty-four hours of as much as 27 gm. The loss of phosphorus, while decided, was not so remarkable as the loss of nitrogen. It was probably over 2 gm.

Such tremendous tissue destruction is a condition that is comparatively rare in other diseases, but it seems to be very common in acute leukæmia, as far as the very few chemical investigations of this disease that are on record indicate. It appears, therefore, to be a very important element in the pathology of the condition. Magnus-Levy, in particular, directed attention to this very remarkable disturbance of metabolism, reporting his own results in three cases, in one of which he determined the nitrogen of the urine, and in the other two the actual balance between the intake and the outgo. In the first of his cases there was a loss in forty hours by the urine alone of 24.8 gm. more than the patient had taken in his food. In addition to that, he lost 3 gm. in the feces and about 17 gm. by hemorrhage, so that in all this patient showed in a period of forty hours a negative balance of nearly 45 gm. of nitrogen. In the second case 85.4 gm. of nitrogen were lost in seven days, and on the last day there was a negative balance of 20.8 gm. In the third case, with a very low food intake, the urinary nitrogen averaged 21 gm. during the last seven days, and on one day the nitrogen excretion in the urine rose to 27 gm.

A similarly marked loss was evidently present in Ebstein's case, in which over 62 gm. of urea were found in the twenty-four hours' urine, and a number of other authors have seen much the same state of affairs. It is of interest that in Wey's case of chronic leukæmia, with a terminal very acute stage, the patient passed per day as much as 29 gm. of nitrogen in the urine. This violent disturbance is apparently not an essential part of the pathology of acute leukæmia, as there are one or two reliable cases on record that showed no marked loss; nevertheless, it appears from the observations that have been made to be a very pronounced and common feature.

Since leukæmia is a disease chiefly of the lymphatic glands and bone-marrow, it is but natural that the metabolism of the nucleins should show most striking evidence of disturbance. The excretion of uric acid and phosphates has been found very excessive in most cases, and some of the figures obtained by Magnus-Levy were exceedingly remarkable. One of his cases showed a loss of about 15 gm.

of P_2O_5 in fifteen hours, a most astonishing figure; and in the same case the uric acid excretion reached 12.22 gm., which, considering this patient's low food intake, was certainly twenty times the normal amount, while the purin bases were 0.321 gm. Similar, though less striking, losses have been observed by others. Wende, for example, reported a case in which the uric acid excretion reached nearly 5 gm. There are, however, a few cases on record in which the uric acid excretion was very slightly, if at all, increased.

It is a matter of great clinical interest, as well as pathological, to attempt to determine the cause of this profound disturbance of metabolism. Magnus-Lævy, because of his own observations on nitrogen metabolism in hemorrhagic disorders, particularly in a case of purpura hemorrhagica, and because of a few observations of other investigators on the relation of hemorrhage to destructive changes in metabolism, reaches the conclusion that a large part, at any rate, of the tissue destruction in acute leukæmia is probably dependent upon the hemorrhages that occur in this disease. This seems to me to be very questionable. Magnus-Lævy's work, and that to which he refers, has shown to be sure that hemorrhagic conditions are often associated with a marked nitrogen loss, and it is not improbable that a small amount of tissue destruction is dependent upon the hemorrhage itself. The experimental work of Hawk and Gies, for example, in which the only abnormality present was loss of blood, shows that mere hemorrhage does produce some tissue destruction. But in such experimental work, in which other abnormalities are not present, hemorrhage seems to cause but slight disorder of general metabolism, and hence it would appear to be an insufficient explanation of the extremely unusual conditions found in acute leukæmia. Furthermore, there are many cases of extensive purpura in which, even in the absence of accurate chemical investigations, the clinical appearances are such as to indicate pretty clearly that there cannot be in all cases a marked tissue loss. Therefore, since very severe abnormalities, besides the hemorrhage, are present in acute leukæmia, it seems to me that hemorrhage offers a very incomplete explanation of the metabolic disturbance in this disease. Violent acute disturbance of metabolism is usually due to some form of toxæmia, and it would appear more rational to look to such a cause in acute leukæmia than to attribute the major portion of the disturbance to hemorrhage; in other words, I should be prepared to learn that the hemorrhage and the disorder of metabolism were due to a common cause, probably a toxæmia.

The conditions in the case of purpura hemorrhagica that I shall at once mention indicate very strongly, I think, that while some tissue destruction occurred in this case also, hemorrhage was not the cause of it; and hence I believe that this case goes far to remove the basis of Magnus-Lævy's hypothesis that hemorrhage is the main cause of the tissue destruction in acute leukæmia.

This patient was a boy aged sixteen years; he was admitted to the University Hospital on Dr. Musser's service December 15, 1904. Admission No. 3531. His trouble had begun eleven days before, with moderate joint pains, which had continued, but had not been severe at any time. There had been little swelling of the joints. Nine days before he was admitted to the hospital hemorrhagic spots had appeared in his skin; these had rapidly increased two days before his admission, and he had also had nausea and vomiting and cramping abdominal pain. The symptoms had become still worse on the following day. When admitted he looked very ill. He had nausea, frequent vomiting, and bloody bowel movements, and showed extensive subcutaneous hemorrhages, particularly on the arms and back. The symptoms continued severe, and on the 20th he was greatly prostrated and delirious, and his hemorrhages had increased to a most striking extent. I have never seen more widespread subcutaneous hemorrhages; they involved almost all the surface of the back, the arms, and much of the forearms; there were large extravasations in the skin over the front of the chest and abdomen, many large patches on the legs and a number on the face. There were also small hemorrhages into the conjunctiva; he had hemorrhages from the mouth and nose; he vomited some blood, and he also passed blood from the bowel and with the urine. I began with this day (*i. e.*, urine of 20th and 21st, marked 21st in table) observations of his excretion of nitrogen, phosphates, and uric acid. The excretion on the first two days represents total loss, as the patient was taking no food at this time; he was, indeed, losing more than is indicated by the urine, as he was vomiting almost constantly. It was also impossible on the 27th and 28th to estimate the intake accurately, as he had severe vomiting much of the time.

It should be noted that on December 21st he was still having hemorrhages, was vomiting violently, and was generally prostrated; on the 22d he was still very ill, but his gastric symptoms had, temporarily, largely subsided, and hemorrhage had, so far as could be determined, stopped entirely; on the 23d nearly all marked symptoms had ceased, and on the 24th he felt so well that I temporarily stopped my observations, as his attack seemed to be over. The point of chief interest is, however, that on the evening of the 24th he had a return of his gastric symptoms, and on the 26th the joint pains, abdominal pain, gastric symptoms, and prostration returned, but he had no discoverable return of hemorrhage. Observations of metabolism were again begun on this day. These symptoms lasted two days, and then decreased. Subsequently, though he had the signs of an acute nephritis, he improved with great rapidity in his general condition, and gained eleven pounds in a week. The figures for January 3d and 4th, therefore, represent his condition during convalescence. At this time he was getting between 11 and

12 gm. of nitrogen in his food. In the preceding periods, when he took food at all, he was probably getting at most 3 or 4 gm. of nitrogen.

Date.	Urine.	Nitrogen.	Uric acid.	P_2O_5.
December 21	1800	22.478	0.196	6.192
" 22	1700	23.738	Lost	4.012
" 23	1350	18.295	0.178	2.484
" 24	870	14.323	0.158	1.581
Return of symptoms, but no hemorrhage :				
December 27	1560	22,579	.....	2.280
" 28	1470	20.580	.....	1.705
Convalescent :				
January 3	880	6.718	.....	0.950
" 4	1275	6.642	.....	0.918

For the first two days these figures show, as far as the nitrogen and P_2O_5 are concerned, conditions that are very similar to those in my case of acute leukæmia, and in the majority of the other cases in which metabolism has been observed, namely, a profound loss. The next day, when there was no hemorrhage and the symptoms were all disappearing, there was a very marked decrease in the nitrogen and P_2O_5 loss; while on the fourth day the conditions were approaching the normal. The first four days, therefore, apparently support Magnus-Levy's view that the hemorrhage may be responsible for much of the metabolic disturbance. The conditions on the 27th and 28th, however, indicate directly the contrary. At this period hemorrhage was absent, but the renewed metabolic disturbance was only slightly less severe than that at the time when hemorrhage was occurring in wide areas, from the mucous membranes and into the skin. The conditions on January 3d and 4th are those to be expected in a convalescent; the man was, as his weight indicated, showing a marked retention, and he was evidently using this in building up tissue.

One case does not, of course, prove or disprove an hypothesis. Nevertheless, the conditions in this case; the fact that extensive hemorrhage often occurs without the clinical appearance of tissue loss; the knowledge that hemorrhage does not necessarily occur in acute leukæmia; and the fact that, besides the hemorrhages, evidences of grave disorders of other kinds are present in acute leukæmia and in many other hemorrhagic cases: all these unite, I think, to make it appear extremely improbable that the tissue loss should be attributed in considerable extent to the hemorrhage, and indicate, on the contrary, that the cause of both hemorrhage and tissue loss must be sought together in the nature of the disturbance that produces the disease.

At the time that I observed my case of acute leukæmia I suggested that it is probable that the tremendous tissue loss is due to some

pathological disturbance of autolytic processes. This suggestion was at that time almost a pure hypothesis, but the hypothesis was based upon the fact that similar metabolic conditions are seen in pneumonia at the time of the crisis, and in acute yellow atrophy of the liver; and in these diseases sudden and very extensive autolytic tissue destruction occurs; in the lung exudate in the one case, in the liver in the other. Since that time further experiment has led physiologists and pathologists to a general acceptance of the importance of autolysis in a great number of physiological and pathological conditions, and the suggestion that the metabolic disturbance in acute leukæmia is a disturbance of autolysis is on general grounds now better justified.

More direct evidence in favor of this view is found, I think, in observations that I have recently made on the influence of the *x*-ray on metabolism in chronic leukæmia.[1] In a case successfully treated there was, immediately upon the use of the *x*-ray, a most astonishing increase in the excretion of nitrogen, uric acid, purin bases, and P_2O_5. The excretion was, on the whole, fully doubled and that of uric acid, purin bases, and P_2O_5 was ultimately much more than doubled. On the other hand, a case of chronic leukæmia that was unsuccessfully treated with the *x*-ray showed almost no change in metabolism as a result of this treatment, and soon died, with constantly decreasing excretion. The fact that normal persons respond to *x*-rays by increase of metabolism, and that the leukæmic successfully treated showed even more striking response, while, on the other hand, the leukæmic unsuccessfully treated, and also two persons with gout had no noteworthy alterations in metabolism as a result of *x*-ray exposures, led to the conclusion that the changes in metabolism that occurred as a result of exposure to *x*-rays are due to some response in the individual and not to any direct physical or other effect of the *x*-ray upon the tissues. The great rapidity of action of the *x*-ray upon metabolic processes, together with the other facts already mentioned, makes it seem highly probable that *x*-rays act in some way in increasing the ferment-like metabolic processes that are normal in the body; in other words, in stimulating autolysis. This becomes almost a definite fact when one considers that Neuberg has shown experimentally that radium increases autolysis *in vitro*.

The conditions in the case of chronic leukæmia that was successfully treated with *x*-rays bore a striking resemblance to the conditions in acute leukæmia, though they were not so extremely pronounced. The fact that autolytic processes can apparently be thus—*i. e.*, under the influence of the *x*-ray—changed from their normal state to one in which there is very unduly rapid tissue

[1] Reported, with Dr. Musser, in the University of Pennsylvania Medical Bulletin, September, 1905.

destruction suggests strongly, I think, that the conditions in acute leukæmia are actually due to some cause that similarly, though more markedly, excites an increase of autolytic tissue destruction.

Until the observations on the influence of the *x*-ray on metabolism in leukæmia were made I was naturally inclined to believe that the conditions in acute leukæmia are a part of the malignancy of the disease. The effect of the *x*-ray on chronic leukæmia, however, suggests strongly that the tissue destruction in acute leukæmia may be not an essentially malignant process, but one that is of benign purpose. In chronic leukæmia *x*-ray exposures produce, in cases successfully treated, a remarkably rapid tissue destruction that is evidently, from the clinical course of these cases, benign in its influence upon the organism. It seems wholly possible, therefore, that the destruction that occurs in acute leukæmia is a response of the organism to the leukæmic tissue hyperplasia, and is an attempt to control this and to destroy the leukæmic tissue. It is not improbable, however, that if this is the case metabolic processes run amuck in the destruction of leukæmic tissue, and carry the destruction so far that other tissues are involved to a dangerous degree; and, too, death is probably often hastened by the intoxication that results from the products of the rapid tissue destruction.

The fact that tissue destruction has not been found greatly increased in all cases of acute leukæmia, but is so in most instances, tends rather to emphasize than to disprove this suggestion that the tissue destruction is an attempt at reaction on the part of the diseased individual, and that the purpose of the reaction is the destruction of diseased tissue. The fact that it is not constant indicates that the severity of the disease is not chiefly dependent upon the tissue destruction, and this latter point may readily be brought in harmony with the view that the tissue destruction is a part of the fight of the organism against the disease. Considering, again, the analogy between acute leukæmia and chronic leukæmia, when the latter is being treated by *x*-rays, the above suggestion concerning the purpose of the tissue destruction in acute leukæmia becomes more emphatic when we remember that the tissue destruction in chronic leukæmia that results from *x*-ray treatment is limited almost entirely, perhaps entirely, to the lymphatic tissues and bone-marrow—that is, to the diseased tissues.

In conclusion, I would refer to the curiously low excretion of uric acid in the case of purpura hemorrhagica, which is of interest when contrasted with the large loss of nitrogen, and particularly with the extremely large amount of P_2O_5 lost. As will be noticed, on the day when hemorrhage was continuing the P_2O_5 excretion was above 6 gm., and on the next day it was still about 4 gm.; amounts that are very excessive at best, and that are particularly so in this instance, because there was no intake of P_2O_5.

Indeed, a noteworthy amount more than this was probably excreted through his vomit. On the other hand, the uric acid figures are about normal for a person who is temporarily taking no food; in fact, they are even rather low for such circumstances. The sources of the P_2O_5 are chiefly bone, nucleins, and other parts of tissues, such as lecithin, that contain much phosphorus. That it came in this instance largely from the bones is, of course, very improbable at best, and is practically ruled out by the fact that the general nitrogen excretion was so great that it indicates marked destruction of nitrogenous tissue. It is, of course, possible that lecithin destruction, which, like bone destruction, would yield no uric acid, was responsible for the excessive P_2O_5 excretion, but it is very difficult to conceive of a process that would cause such an extensive destruction of lecithin and of nitrogenous tissues in general and leave the nucleins uninvolved. It seems far more probable that, as is usually the case, it came from nuclein, and that uric acid had been produced; but that, owing to some peculiarity in the disease or the individual, in this case the uric acid had been destroyed and was probably excreted as urea; while in most of the acute leukæmia cases that have been studied the uric acid was not further changed, but was excreted as such. The observations of Magnus-Levy and others on hemorrhagic diseases did not include estimations of the uric acid and P_2O_5, so that I cannot state whether the conditions that I found are frequent in purpura and other hemorrhagic conditions, or were merely peculiarities of this individual case. It would be of interest to determine whether this is a common feature in purpura hemorrhagica as contrasted with other hemorrhagic conditions. If so, it would show that the metabolic disturbances of acute leukæmia and purpura hemorrhagica, though they may closely resemble each other in a gross manner, may exhibit some striking differences in details. It would then be further evidence of the fact that has long been so evident, both clinically and pathologically, that hemorrhagic conditions of various sorts are very different in their natures, though the superficial resemblance provided by the hemorrhages leads, in a clinical discussion, to the inclusion of a number of those that are least understood under one general heading.

Literature.

Magnus-Levy. Virchow's Archiv, Bd. clii.
Ebstein. Deutsch. Archiv f. klin. Med., Bd. xliv.
Wey. Ibid., Bd. lvii.
Wende. The American Journal of the Medical Sciences, December, 1901.
Hawk and Gies. American Journal of Physiology, June, 1904.

SOME OBSERVATIONS ON THE EFFECT OF CERTAIN DIET CURES IN DIABETES MELLITUS.

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"In no other disease is the diet of greater importance than in diabetes. It may be said that there is no rational treatment for diabetes other than the dietetic. When the physician and patient are wearied and discouraged by the great duration of this disease they seek recourse to water cures and bathing resorts, as well as aid from drugs, old and new, of animal, vegetable, or mineral origin. Such measures can only be followed as side issues, and even then can only be practised with great danger, even for a short period of time; for in diet alone do we possess the actual remedy which fulfils the indications for treatment, namely, the maintenance of nutrition, the increase in the ability of the body to assimilate sugar and the avoidance of complications" (von Noorden).

It is not our intention to attempt to cover the field of the dietetic treatment of diabetes in this paper. We wish, however, to bring forward certain diet cures which have assumed some importance during the past few years. These are three in number: the milk cure, the potato cure, and the oatmeal cure.

Milk Cure. According to Naunyn,[1] the merits of a strict milk diet in certain cases of diabetes have been recognized for some years. Rollo advised its use in quantities of a litre a day, and Donkin[2] considered it a valuable food in the treatment of this disease, believing in its efficacy in many cases in reducing and even causing the sugar to disappear entirely from the urine. Winternitz and Strasser[3] and Kolisch[4] have more recently attempted to emphasize the truth of this observation, believing that a strict milk diet will either diminish or even cause the glycosuria to disappear entirely in from twenty-four to forty-eight hours in most cases of this disease. A certain number of patients are entirely relieved by means of the milk cure and remain so after again being placed on a diet containing a considerable amount of carbohydrate material. Naunyn points out that even in severe forms of diabetes ½ a litre of milk a day is often well borne without increasing the glycosuria. He observes, however, that diabetic patients react differently to milk-sugar, and that a trial must, therefore, be made in each individual case. He also draws attention to the fact that often the

unfavorable effect of milk can be detected only after a lengthy and continuous use of this article of food.

A. Berger[5] also studied the effect of a strict milk diet in diabetic glycosuria. He showed that a patient who was entirely free from sugar in the urine on taking 50 grams of bread and ¼ of a litre of milk a day, passed 3 grams of sugar after taking 3 litres of milk in the same length of time. Another diabetic patient who excreted 64 grams of sugar after taking four rolls and 2 litres of milk, produced but a few grams of sugar on a strict milk diet of 2 to 3 litres. Hirschfeld,[6] Blumenthal,[7] Lenné,[8] and von Noorden[9] all believe that it is impossible to formulate any definite rules regarding the effect of milk in diabetes, but consider that every case must be tested individually to determine the tolerance for this particular article of food.

According to von Noorden, milk should be never given in mild forms of diabetes in which glycosuria appears on the taking of this food. Von Noorden also admonishes against the use of a strict milk cure in all cases of diabetes, even when glycosuria does not appear after taking as much as 4 litres of milk daily, as this form of food frequently produces an intense anorexia which is often very difficult to overcome. He advises that in those cases of diabetes in which glycosuria is absent on a milk diet to vary the milk with kefir, buttermilk, kumyss, and cream. In order to avoid any deleterious effect of the milk-sugar, von Noorden recommends the use of the so-called diabetic milk (Gärtner's process), from which a large portion of the milk-sugar has been removed. This product contains but 1 per cent. of milk-sugar.

Hutchison[10] advises the following method for preparing diabetic milk, which contains but a trace of sugar:

Sugar-free Milk for Diabetic Feeding. Take 1 litre of skim milk, heat to a temperature of 30° C., and add 10 c.c. of glacial acetic acid, diluted with 100 c.c. of water. Mix and allow the mixture to stand for about fifteen minutes. Collect the separated casein, and let it drain on very fine muslin, using no pressure. Remove the casein to a mortar, rub into a smooth paste, add ½ litre of distilled water, and strain as before. Repeat this washing of the casein twice. Transfer to a mortar, rub until quite smooth, and add 2½ grams of potassium hydrate dissolved in 100 c.c. of water (or as much of the potassium hydrate as is necessary to make the product just alkaline to phenolphthalein). Add 100 grams of ordinary Devonshire clotted cream, 5 grams of gelatin previously dissolved, 0.06 grams (1 gr.) of saccharine and water, at about 38° C., up to 1 litre. Lastly, strain through fine muslin.

Our own observations with the use of milk as a form of diet in diabetes are in accord with those of von Noorden and other recent writers. We have rarely found it advisable to place any of our patients on an exclusive milk diet, except in those severe cases

of diabetes in which diacetic acid is present in the urine and in which the patient is threatened with the onset of diabetic coma or in which this condition had already set in. On the other hand, we have utilized from ½ to a litre of milk daily in many of our cases of diabetes in addition to other allowable foods, often with excellent results. It is important, however, as has already been pointed out, to observe the effects of this form of food, lest untoward effects be produced and great injury be done the patient. The following cases illustrate the effect of a milk diet in diabetic cases:

CASE I.—A. B., a male, aged fifty-eight years; the patient has had diabetes for about a year; he has lost ten pounds in weight. On an unrestricted diet he passed as much as 3.9 per cent. of sugar in his urine (116 grams daily). When placed on a diet free of carbohydrates (von Noorden's standard diet) his urine became free of sugar; which was still the case when as much as 150 grams of bread were added to this diet. This case can therefore be considered a mild form of the disease. This patient could consume a litre of milk a day without producing glycosuria.

On the other hand, Case II., J. T., female, aged forty-four years, with conditions almost identical with Case I., showed a far less degree of tolerance for milk. This patient passed 2.8 of sugar (68 grams per day) on an unrestricted diet; and no sugar, either on a von Noorden's standard diet or on this diet, with the addition of 150 grams of bread; yet on the addition of ½ litre of milk, 1.8 per cent. of sugar (40 grams per day) was eliminated, and on 1 litre, 2.5 per cent. (58 grams).

CASE III.—E. K., a male, aged forty-two years, illustrates the effect of milk in a severe form of diabetes. This patient had lost forty-four pounds in flesh, and on an unrestricted diet passed 4.8 per cent. of sugar (148 grams daily); on a von Noorden's standard diet, 3.2 per cent. of sugar (83 grams daily). His urine, even under these conditions, presented considerable quantities of diacetic acid. When placed upon an exclusive milk diet for ten days, the percentage of sugar decreased to 2.1 per cent. (42 grams daily), the diacetic acid disappeared, and he began to increase slightly in flesh.

CASE I.—A. B., male, aged fifty-eight years; duration of disease one year; former weight 148 pounds.

Observation.	Diet per day.	Daily quantity of urine c.c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Present weight.
1	Unlimited . . .	2980	1037	3.9	116	None	138 lbs.
2	Standard diet (von Noorden)	2010	1026	None	None	"	
3	Standard diet + 150 gm. bread	2020	1028	"	"	"	
4	Standard diet + 150 gm. bread + 1 litre milk	2000	1028	"	"	"	

CASE II.—J. T., female, aged forty-four years; duration of disease six months; former weight 122 pounds.

Observation.	Diet per day.	Daily quantity of urine c.c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Present weight.
1	Unlimited diet . .	2450	1032	2.8	68	None	116 lbs.
2	Standard diet . .	2000	1026	None	None	"	
3	Standard diet + 150 gm. bread	2000	1024	"	"	"	
4	Standard diet + 150 gm. bread + ½ litre milk	2280	1039	1.8	40	"	
5	Standard diet + 150 gm. bread + 1 litre milk	2350	1030	2.5	58	"	

CASE III.—E. K., male, aged forty-two years; duration of disease three years; former weight 188 pounds.

Observation.	Diet per day.	Daily quantity of urine c.c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Present weight.
1	Unlimited diet . .	3100	1040	4.8	148	Present	144 lbs.
2	Standard diet . .	2600	1036	3.2	83	"	
3	Milk diet (3 litres) .	2000	1030	2.1	42	None	

POTATO CURE.—Mossé[11] first advised the potato cure as a means of treating patients suffering with diabetes. His plan is to replace all carbohydrates by potatoes for a period of several weeks. Patients are to receive 1500 grams of potatoes, replacing 500 grams of wheat bread in as much as according to his experiments patients could tolerate from two and one-half to three times as much of potatoes (weighed raw) as they could of wheat bread. Potatoes are relatively poor in carbohydrates containing from 16 to 22 per cent. of starch, while wheat bread contains about 60 per cent. During the time of the potato treatment no other carbohydrates are to be consumed. Mossé found a marked diminution in the glycosuria and polyuria while the patient was taking the potato starch, as well as a diminution in thirst and an increase in strength. He also observed that a certain degree of tolerance for wheat bread was produced after the patient had been on the potato cure for several days. He attributes the beneficial effect of this diet to the large proportion of water and potassium salts contained in the potato. The large quantity of fluid diminishes the thirst, while the potassium salt appears to improve the general nutrition. In 23 diabetic patients to whom diets of bread and potato were alternately given, an advantage in favor of the bread was found in only 1 case.

Offer[12] has confirmed the observation of Mossé, and von Noorden,[13] too, extols this plan of treatment, and recommends that the potato need not only be given as the only carbohydrate food in

diabetes, but also that it may be utilized to replace a certain portion of the bread; for instance, in a diet in which 75 to 125 grams of wheat bread are allowable an amount of potato represented by a third of this quantity can be substituted for it; so that in this instance 75 to 125 grams of potato can be consumed daily, leaving as much as 50 to 85 grams of bread still to be eaten. In this way the variety of carbohydrate food is increased, and at the same time large quantities of fat can be consumed in the form of butter, which may be added to the potatoes. This is a matter of great importance for inasmuch as a large proportion of the carbohydrates are excluded in this disease, this heat-producing food which is eliminated must be substituted for by some other form of food, and this is best accomplished by means of fats, such as butter.

In our own observations with the use of the potato cure in the treatment of diabetes, we have never followed Mossé's plan, that is, by replacing all carbohydrates by potatoes, but have rather followed the method suggested by von Noorden, replacing a portion of the bread by this form of food. A patient who is permitted to consume 100 grams of bread a day can take 300 grams of potatoes, so that at least a certain proportion of the bread can be replaced by this form of food. If care be taken to regulate these proportions we have never seen the slightest harmful effect produced by the use of potatoes, even in severe forms of diabetes. In a not inconsiderable number of cases very beneficial results were obtained by us. The potato has an additional advantage, in that it lends variety in furnishing carbohydrate food in as much as it can be prepared in various ways, baked, boiled, fried, mashed, etc., and again as being a means, as has already been pointed out, through which fatty food, such as butter, can be furnished the body in rather large quantities. The following cases illustrate the effect of a potato diet in various forms of diabetes:

CASE IV.—J. H., a male, aged forty-nine years, who had suffered with diabetes for nine months and who had lost twenty-two pounds in weight, illustrates how the potato diet acts in a mild form of diabetes. This patient who passed 4.1 per cent. of sugar in his urine (115 grams daily) on an unrestricted diet was free of all sugar in his urine on a standard diet, and also when 150 grams of bread were added to the diet; the absence of sugar continued when to this diet 50 grams of bread and 300 grams of potatoes were added. This patient passed about 1 per cent. of sugar (20 grams daily) on the usual diabetic diet, with the addition of 60 grams of bread, but when only 20 grams of bread were allowed and 120 grams of potatoes he passed but 0.9 per cent. of sugar in his urine (18 grams daily).

CASE V. illustrates how the potato diet acts in a moderately severe form of diabetes. This patient, a female, aged sixty-two years, had had diabetes for two years; she had lost twenty-eight pounds in weight; on an unlimited diet she passed 4.6 per cent. of sugar (138 grams daily; on a standard diet, none; on a standard diet,

with the addition of 60 grams of bread, 2.4 per cent. (67 grams daily); on a standard diet, with 20 grams of bread and 120 grams of potatoes, but 2.1 per cent. (54 grams daily); on the usual diabetic diet, with the addition of 60 grams of bread, 3.2 per cent. (83 grams daily); while on the same diet, with but 20 grams of bread and 120 grams of potatoes, but 2.8 per cent. (73 grams daily).

CASE VI. illustrates the effect of the potato diet on a severe form of diabetes. This patient was a female, aged thirty-seven years, who had been suffering with the disease for three years.

On an unlimited diet the patient passed as much as 5.1 per cent. of sugar (163 grams daily); on a von Noorden standard diet this amount was reduced to 3.6 per cent. (90 grams daily); when to this standard diet 60 grams of bread were added, the patient passed 4.5 per cent. of sugar (135 grams daily), which was reduced to 4 per cent. (112 grams daily); when one-third of the bread was replaced by a proportionate amount of potato. On the usual diabetic diet containing 60 grams of bread, this patient passed 4.8 per cent. (144 grams daily) of sugar, which was reduced to 4.6 per cent. (133 grams daily), when 40 grams of the bread were replaced by 120 grams of potato.

CASE IV.—J. H., male, aged forty-nine years; duration of disease nine months; former weight 180 pounds.

Observation.	Diet per day.	Daily quantity of urine c c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Present weight.
1	Unlimited diet . .	2800	1034	4.1	115	None	162 lbs.
2	Standard diet . .	2050	1026	None	None	"	
3	Standard diet + 150 gm. bread	2120	1026	"	"	"	
4	Standard diet + 50 gm. bread + 30 gm. potato	2020	1028	"	"	"	
5	Usual diabetic diet + 60 gm. bread	2140	1028	1.0	20	"	
6	Usual diabetic diet + 20 gm. bread + 120 gm. potato.	2020	1024	0.9	18	"	

CASE V.—Z. L., female, aged sixty-two years; duration of disease two years; former weight 162 pounds.

Observation.	Diet per day.	Daily quantity of urine c.c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Present weight.
1	Unlimited diet . .	3000	1038	4.6	138	None	134 lbs.
2	Standard diet . .	2060	1024	None	None	"	
3	Standard diet + 60 gm. bread	2800	1032	2.4	67	"	
4	Standard diet + 20 gm. bread + 120 gm. potato	2600	1030	2.1	54	"	
5	Usual diabetic diet + 60 gm. bread	2620	1032	3.2	83	"	
6	Usual diabetic diet + 20 gm. bread + 120 gm. potato.	2610	1032	2.8	73	"	

CASE VI.—M. N., female, aged thirty-seven years; duration of disease three years; former weight 172 pounds.

Observation.	Diet per day.	Daily quantity of urine c.c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Present weight.
1	Unlimited diet . .	3200	1040	5.1	163	None	139 lbs.
2	Standard diet . .	2500	1034	3.6	90	"	
3	Standard diet + 60 gm. bread	3000	1036	4.5	135	"	
4	Standard diet + 20 gm. bread + 120 gm. potato	2800	1035	4.0	112	"	
5	Usual diabetic diet + 60 gm. bread	3000	1038	4.8	144	"	
6	Usual diabetic diet + 20 gm. bread + 120 gm. potato	2900	1036	4.6	133	"	

OATMEAL CURE. Von Noorden[14] first pointed out that certain patients suffering with diabetes, who, notwithstanding the fact of being on a strict diet, continued to eliminate sugar, could at times be quickly and permanently relieved of this condition by being placed upon a diet of large quantities of oatmeal. He also pointed out that in many severe forms of diabetes oatmeal is twice as well borne as an equivalent quantity of bread, and frequently by gradually increasing the quantity of oatmeal the glycosuria did not increase, while acetone and diacetic acid disappeared. The beneficial results were, however, only obtained when the oatmeal represented the largest proportion of food consumed, when no other carbohydrates were taken and no meat eaten. Von Noorden advises that the oatmeal cure should be undertaken for eight to fourteen days only, inasmuch as the patient will become satiated with this article of food after this length of time. In some instances, after a return to the usual diet, the tolerance for carbohydrates increases, while in others it does not, and glycosuria rapidly returns, and frequently diacetic acid, too.

In a more recent publication von Noorden[15] reports the results of his observations in 100 cases of diabetes treated by means of the oatmeal diet.

The results are classified into five classes:

1. Those cases in which it was impossible to produce a glycosuria, notwithstanding a strict diet. In these instances remarkable results were frequently obtained by means of the oatmeal cure, which could not have been obtained in any other way. It was often possible in these cases to allow a certain proportion of carbohydrates in addition to the usual diabetic diet without producing glycosuria.

2. In another class of cases good results were obtained from the oatmeal cure, the results were not permanent, although by means of this form of treatment the onset of diabetic coma was prevented.

3. In another class the tolerance for carbohydrates did not increase

after inaugurating the oatmeal cure, but aceton diminished and remained at a minimum, even after a return to the usual diet.

4. In not an inconsiderable proportion of cases the glycosuria increased rather than diminished on the oatmeal diet.

5. In mild forms of diabetes without diaceturia the oatmeal diet was badly borne.

In a general way it may be stated that the oatmeal cure is especially useful in those forms of diabetes exhibiting diacetic acid in the urine and that in mild forms it is not only a useless form of treatment, but may even prove harmful. On the other hand, as von Noorden points out, each case of diabetes should be studied individually and daily examinations of the urine should be made, in order to determine whether the special case is being benefited by this form of treatment. In a recent communication, Mohr,[16] working under von Noorden's direction, shows that the oatmeal cure is especially useful as a means of diminishing the acetone in diabetic cases.

In diabetic coma von Noorden recommends the oatmeal cure as preferable to the milk cure, inasmuch as it usually diminishes the aceton bodies more quickly. He believes the beneficial effect of the oatmeal is due to the substances contained therein, especially the salts; by analysis of the feces he showed that the lessened excretion of sugar in the urine was not due to a lessened absorption of the oatmeal in the intestines.

Von Noorden's directions for preparing the oatmeal are as follows: Knorr's oatmeal or Hohenlohe's Haferflocken (but any good oatmeal may be used) is well cooked for a considerable length of time in water with the addition of a bit of salt; while cooking butter and a vegetable albumin are added or the white of egg beaten and strained may be mixed with it, as the oatmeal cools. Roborat or a rice albumin (Bremer Brotfabrik) is recommended as being especially palatable mixed with the oatmeal. The quantities to be taken daily are: oatmeal, 250 grams; albumin, 100 grams; butter, 300 grams.

The meals are taken at two-hour intervals; in addition, von Noorden permits cognac or wine and some strong black coffee; after a few days five to seven eggs, but no meat; after eight to fourteen days a vegetable diet day is interposed and occasionally at rare intervals, if the oatmeal diet is to be kept up for any length of time, a small quantity of meat or fish is allowed to relieve the monotony of the diet and render it bearable. The return to the ordinary diet must be made cautiously, lest the acetone bodies increase to an alarming degree.

Our results with the oatmeal diet have been very satisfactory. In mild forms of diabetes this treatment should never be utilized. This is well illustrated in Case VII. The patient, a man, aged sixty-two years, had suffered with diabetes for six months; on an

unrestricted diet he passed 2.2 per cent. of sugar in his urine (48 grams daily); on a standard diet the urine became sugar-free, which was also the case when 150 grams of bread were added to the standard diet; on an oatmeal diet the patient passed as much as 4 per cent. of sugar (112 grams daily), which was reduced to 1 per cent. (24 grams daily) when the patient was placed on the usual diabetic diet.

The oatmeal cure has its place only in the severe forms of diabetes. In these cases we have obtained remarkable results. In certain cases it is possible to rid the urine of sugar entirely when this is impossible on an entirely carbohydrate-free diet (standard diet).

Case VIII. illustrates this condition. This patient, a female, aged forty-four years, had had diabetes for eight years; on an unrestricted diet she passed 5.8 per cent. of sugar (185 grams daily), which was reduced to 3.1 per cent. (86 grams daily) on a standard carbohydrate-free diet. This patient was placed on the oatmeal diet for eight days, and the sugar disappeared entirely from the urine; it reappeared when the patient was placed on the usual diabetic diet, but never reached such large proportions as it did originally.

In another class of cases the oatmeal diet not only occasions a disappearance of the sugar in the urine, but also the diacetic acid. The condition is represented in Case IX., F. F., a female, aged fifty-nine years, who had had diabetes for four years; on an unrestricted diet she passed 6.8 per cent. of sugar (250 grams daily); on the usual diabetic diet 5.4 per cent. (180 grams daily); after being placed on an oatmeal diet for eight days both sugar and diacetic acid disappeared. While sugar reappeared in the urine after the patient had been placed on the usual diabetic diet, it did not reach the large percentage it had gotten to before, nor did diacetic acid reappear.

Finally, there are a number of cases, of which Case X. is a representative, in which, while the percentage of sugar present in the urine is not materially affected by the oatmeal cure, diacetic acid disappears under its influence and remains absent. Thus, Case X., K. C., female, aged thirty-eight years, who had had diabetes for two years, passed 6.2 per cent. of sugar (248 grams daily) when taking an unrestricted diet; on the usual diabetic diet, this amount was reduced to 5.5 per cent. (176 grams daily). The percentage of sugar remained at 5 per cent. (170 grams daily), even after the oatmeal cure had been inaugurated; but whereas diacetic acid had been present while this patient had been on an unrestricted diet, as well as a diabetic diet, it disappeared entirely after the patient had been taking the oatmeal for a few days.

In conclusion, we believe we are justified in asserting that the milk cure, potato cure, and oatmeal cure are valuable aids in the treatment of diabetes.

Great care, however, must be practised with the use of these various forms of diet. Each case must be studied individually and must be carefully watched in order to obtain the very best results.

CASE VII.—J. L., male, aged sixty-two years; duration of disease six months; former weight 186 pounds.

Observation.	Diet per day.	Daily quantity of urine c c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Weight.
1	Unlimited diet . .	2200	1028	2.2	48	None	175 lbs.
2	Standard diet . .	1800	1026	None	None	"	
3	Standard diet + 150 gm. bread	2000	1026	"	"	"	
4	Oatmeal diet . .	2800	1038	4.0	112	"	
5	Ordinary diabetic diet	2400	1026	1.0	24	"	

CASE VIII.—T. S., female, aged forty-four years; duration of disease eight years; former weight 172 pounds.

Observation.	Diet per day.	Daily quantity of urine c.c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Weight.
1	Unlimited diet . .	3200	1040	5.8	185	None	124 lbs.
2	Standard diet . .	2800	1032	3.1	86	"	
3	Oatmeal diet . .	2000	1024	None	None	"	

CASE IX.—F. F., female, aged fifty-nine years; duration of disease four years; former weight 184 pounds.

Observation.	Diet per day.	Daily quantity of urine c c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Weight.
1	Unlimited diet . .	3800	1042	6.8	250	Marked	140 lbs.
2	Ordinary diabetic diet	3400	1040	5.4	180	"	
3	Oatmeal diet . .	2400	1028	None	None	None	

CASE X.—K. C., female, aged thirty-eight years; duration of disease two years; original weight 130 pounds.

Observation.	Diet per day.	Daily quantity of urine c.c.	Specific gravity.	Percent. of sugar.	Total quantity of sugar per day.	Diacetic acid.	Weight.
1	Unlimited diet . .	4000	1040	6.2	248	Marked	110 lbs.
2	Ordinary diabetic diet	3200	1038	5.5	176	"	
3	Oatmeal diet . .	3400	1036	5.0	170	None	

LITERATURE.

1. Naunyn. Nothnagel's specielle Pathologie u. Therapie, Bd. vii. p. 366.
2. Donkin. On the Relation between Diabetes and Food, 1875.
3. Winternitz and Strasser. Centralblatt f. innere Medizin, 1899, No. 45.
4. Kolisch. Wiener klinische Wochenschrift, 1900, No. 52.
5. A. Berger. Wiener klinische Rundschau, 1900, No. 31.
6. Hirschfeld. Die Zuckerkrankheit., Leipzig, 1902.
7. Blumenthal. Zeitschrift f. Diatetische und Physikalische Therapie, 1899.
8. Lenné. Deut. Medizinalzeitung, 1903, No. 64.
9 von Noorden. Wiener Medizin. Presse, 1902, No. 40, and Die Zuckerkrankheit. und ihre Behandlung. Berlin, 1901.
10. Hutchison. Food and Dietetics, p. 474. See also Friedenwald and Ruhräh, Diet in Health and Disease, p. 613.
11. Mossé. Comptes-rendus, tome cxxxiii., No. 24, and Revue de médecine, 1902, tome xxii.
12. Offer. Discussion at the Gesellschaft f. innere Medizin, Vienna, December 3, 1903.
13. von Noorden. Leyden's Handbuch der Ernährungs Therapie, Bd. xi. p. 244.
14. von Noorden. Wiener Medizin. Presse, 1902, No. 40.
15. von Noorden. Berliner klinische Wochenschrift, 1903, No. 36, p. 817.
16. Mohr. von Noorden's Sammlung klinische Abhandlungen über Pathologie u. Therapie der Stoffwechsel u. Ernährungsstörungen, 1904.

GANGRENE OF THE TONSIL.[1]

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AT the twenty-fifth annual meeting of the American Laryngological Association, held in Washington, I had the pleasure of presenting a case of this unusual condition, which was read by title. Since the report of this case, I have had the fortune to observe another case of a similar character, and, as the condition seems to be of such unusual character, I have incorporated my first case in this paper, as well as given abstracts of the only 3 cases which I find under the title of gangrene of the tonsil in medical literature. It seems passing strange that this condition should be so infrequently observed; more strange, if of such infrequent occurrence, that I should have had 2 cases under my observation within fourteen months. The diagnosis presents no difficulty; the physical signs and the constitutional manifestations are too positive. The exclusive bacteriological investigation and the rapid fatal termination can leave no doubt in the minds of the most skeptical diagnostician. The characteristic stench of the disorganized tissue is, like the odor of atrophic rhinitis, once recognized, never forgotten.

CASE I.—On March 1st I was called in consultation to see a patient residing about nine miles from Washington. It was late in the afternoon when I reached the patient's house, and it was nearly an hour later before the consultants were with me.

On the presentation of the case to me by one of the consultants and carefully going over the history and the temperature sheets, I

[1] Read before the American Laryngological Association, 1905.

could see at once that the case, whatever might be its cause, was one of extreme sepsis. The history as presented to me was as follows:

The patient was a man of robust physique, aged forty-five years, addicted to excesses. About ten days before he had returned from a trip to the North, at which time he had been using alcoholics to excess. When he first came under the observation of the physicians in charge, he had considerable difficulty in swallowing, marked infiltration of the right side of the neck, with other characteristic symptoms of quinsy. In a couple of days it was stated that the patient had a discharge of pus from the nose and mouth, with which there was a subsidence of the swelling in the right side of the neck, a restoration of the normal voice, no further difficulty in deglutition, and the return of the ability to open the mouth freely and widely. About two days thereafter the physician in charge saw what he supposed to be some white spots on the right tonsil. He then thought that the case was probably one of diphtheria, although there was a doubt in his own mind as to the diagnosis. The next day, however, he administered 3000 units of antitoxin. On the second day thereafter there were 3000 more units given. From the first administration until I saw him he received in all 16,000 units of antitoxin. Even under this repeated use of antitoxin there was no improvement in the patient's condition. He gradually but progressively grew worse. On the ninth day of his illness the patient became delirious, and on the tenth and last day of his life I was called in consultation.

At this time the patient's temperature was 102°, his pulse 120, soft and compressible; and he was in a muttering delirium. On inspection, which was comparatively easy for one in a delirium, and in such a serious condition, I was at first struck by the absence of the characteristic diphtheritic odor of the breath, and was impressed by the peculiar odor which is due to sloughing tissue. On depressing the tongue I was quite unprepared for the sight which I observed. On the right side the tonsil, the anterior and the posterior pillar, and as far down in the pharynx as I could see was one continuous mass of grayish, brownish, putty-like slough. The left tonsil and the curtain were perfectly normal. I particularly noticed the fact that there had already formed a complete line of demarcation between the necrotic and the normal tissue. The examination was prolonged until a careful and thorough inspection was made of the diseased area, and both the consultants had examined it with the same care. On consideration of the case, they both told me that the first they had noticed of the sloughing was the day before, when there was a narrow white line of diseased tissue; but, judging from the complete line of separation, I was rather inclined to believe that their examination was not as perfect a one as had been made by me. The patient was *in extremis*, and died two hours after I first saw him in consultation with Drs. Chappil and Kleinschmidt.

CASE II.—On May 17, 1904, I was called in consultation by Dr. Edward F. Pickford to see an unusual case of ulceration of the tonsil. The subject was a man, aged thirty-six years. His occupation was that of an insurance agent. He was married, and, as far as could be ascertained, led a moral life in every respect. Denied the existence or possibility of venereal infection; no past evidence of such infection. Personal habits good. So far as could be elicited, he had led a uniform, regular life, from which there had been no deviation in past six months. Family history good. Had had the usually infectious diseases of childhood. Dr. Pickford told me that he had been called to see the patient on May 4th. The patient stated to him that he had had a sore mouth and sore throat for several days. Mastication and deglutition were very painful and difficult. The patient was very fretful and extremely despondent. The temperature was 99.8°. On inspection the breath was noted to be extremely foul; the tongue heavily coated; the gums congested, swollen, bleeding readily, and separated from the teeth. There were some isolated grayish patches along the lower borders of the gums. The tonsils, particularly the right one, were very much enlarged and tender to the touch, at this time showing no evidences of ulceration. Two days later ulcerative patches were manifest on the right tonsil. By May 12th patient's condition had markedly improved. His temperature was normal; he could eat and drink with comfort, and his depression of spirits had given way to his usual happy demeanor. The swollen gums had receded, the patches on gums and tonsils had disappeared, and the tonsils were much reduced in size. Dr. Pickford discharged himself, but was recalled to the patient May 15th. On this latter visit Dr. Pickford found quite a change in the condition of his patient. The principal lesion was now in the tonsillar region. The left tonsil was covered with a dirty, grayish-looking patch. The mouth was nearly normal. The throat was extremely sore and the patient was markedly irritable and greatly depressed. Temperature, $99\frac{3}{5}°$. As the deposit on the tongue looked diphtheritic, Dr. Pickford made a culture, with negative result. Dr. Pickford told me on our consultation that he thought the case at first to be one of ulcerative stomatitis of scorbutic origin. Examination made by Dr. Pickford of other systems and organs of the body showed them to be in apparent normal condition. When I first saw this patient his countenance showed an exceedingly anxious expression; he was very restive, but not unduly nervous. Complained greatly of difficult breathing, and was excessively despondent and anxious as to his welfare. Told me that his throat pained him greatly; that it was almost impossible to swallow food, and that he feared to assume a recumbent posture for fear that his throat would close. Temperature, $100\frac{1}{5}°$. On examination, under excellent illumination, I found no change in the buccal cavity. There was a redness along the edges of the gums and they seemed

slightly raised from the teeth. On examination of the fauces the change in the right tonsil impressed me at once. The whole surface of the right tonsil was covered with a blackish scab-like exudate, which had an exceedingly offensive odor. There was no change in the left tonsil or in the other portions of the oral pharynx. The blackish exudate was subjected to a most vigorous curetting, nevertheless, without being able to reach healthy tissue. The tissue removed had all the appearances and smell of necrotic tissue. A bacteriological examination had already excluded the presence of the Klebs-Loeffler bacillus. The case was diagnosed as dry gangrene of the tonsil. On May 18th I again saw the patient. His general condition was slightly improved, his temperature the same, although the local condition looked more unfavorable. There was a recurrence of the slough all over the area from which it had been removed the day before, as well as extending to the pillars of the fauces. Again very active curettement was carried out. Smears were then made and placed in the hands of Dr. D. W. Prentiss, who made the following report:

"*Smear from tonsil;* Dr. Charles W. Richardson's case, May 21, 1904. A diplococcus resembling the D. lanceolatus more abundant than any other organism. Staphylococci fairly abundant. No bacilli having the characteristics of the fusiform bacillus of Vincent present. No spirillæ or tubercle bacilli present."

I saw the patient again on May 20th. The necrosis was not only more extensive in depth, but also in area. Some attempt was made to curette away the necrotic tissue at this visit, but the hopelessness of the case, the weakness of the patient, and his pleas caused us to desist. The patient died from exhaustion June 1, 1904.

I shall here incorporate an abstract of the three cases previously reported in the literature:

Abstract I.—The case was that of a negro man, aged forty-five years, who was admitted to the Roosevelt Hospital August 13th. Indications are given in the history, which is quite full, of a sore throat first noticed on the 18th. The patient was rebellious against the use of the depressor, and little was seen in the pharynx. Note of August 20th recites the death of the patient from hemorrhage from mouth and nose. At the autopsy "a large ulcerated area was found in the pharynx occupying the site of the right tonsil and extending for some distance on the postpharyngeal wall; to this a large slough was attached by one extremity. The odor was very offensive."—"Gangrenous Tonsillitis, Fatal Hemorrhage," by E. B. Cragen, *New York Medical Journal*, September 1, 1888, vol. xlviii. p. 233.

Abstract II.—The patient was a young man, aged twenty-six years. Had been attended a week for a sore throat when seen by Dr. Howe. "He had the peculiar pallor and partial stupor or lethargy of considerable toxæmia. The stench in the room was

simply fearful, and at once suggested some necrotic process." At the beginning he had the ordinary symptoms of a peritonsillar abscess. There was a fetid, brownish fluid issuing from his mouth. Deglutition was almost impossible from the great pain; temperature, 104.4°; pulse from 110 to 120. "On looking in his mouth, I found his gums, the inner surface of the labia, and the under surface of his tongue greatly swollen and covered with deep excoriations or ulcers. Both tonsillar organs were swollen and of a deep red or purplish color, indicating considerable stasis. On the left side was a large tumor that involved the entire tonsillar region, extending beyond the median line nearly to the opposite tonsil and well up into the nasopharynx. On the anterior surface of this tumor was a circular necrosed spot about half an inch in diameter, of a peculiar greenish color of gangrenous tissue, and in its centre was a perforation through which came a dark brownish, grumous discharge." The mass, when removed, was black and firm, and could be sliced much as putty or moulding clay can. It had all the appearance of having undergone a dry, gangrenous process. The odor was intense. "The removal of the tonsil exposed a large cavity lined with half an inch or more of greenish-black, necrosed tissue." The treatment was cutting away gangrenous tissue, spraying with hydrogen dioxide and the application of nitric acid. By this procedure the gangrenous tissue was entirely removed and patient recovered in about three weeks. About three months later the patient succumbed to another attack.—Report of a "Case of Gangrene of the Tonsil," by Alexander C. Howe, *Philadelphia Medical Journal*, March 17, 1900, No. 11, vol. v. p. 643.

Abstract III.—The case described by Dr. McVeigh occurred in a girl, aged ten years. The gangrene affected both tonsils. The odor emitted was the characteristic gangrenous stench, which, from his description, must have been very penetrating. The diseased condition had existed for several weeks before coming under his care. The child succumbed one week after being under his observation. He discusses the case, and, from his remarks, we can readily judge that he was alive to the nature of the case and its differentiation from the tonsillar ulceration of scarlatina, diphtheria, measles, and abscess.—"Gangrenous Eschar of the Tonsils, Death," by John Francis McVeigh, *Dublin Quarterly Journal of Medical Science*, 1867, vol. xliv. p. 78.

Remarks. When preparing the report of my first case of gangrene of the tonsil, I had occasion to look up the literature on the subject and was quite surprised at the small number of reported cases. While preparing this paper I again carefully searched the cards of the Surgeon-General's library on the subject of the tonsil, with the result of finding only the three cases which I have above abstracted.

In my first case, on account of the circumstances attendant upon the location of the patient and his rapid death, no satisfactory study

could be made as regarded the exact method of infection. Every effort was made to trace out the source of infection in the second case—the result was as fruitless as that in the first case. The patient was in perfect health at time of infection, giving daily attention to his business. All organs of the body were functionating normal. Neither in his domicile nor in the food ingested could the source of infection be traced. The bacteriological examination does not furnish us with much positive information as to the infecting organism. A diplococcus is not the usual type of organism producing gangrene, yet it is the one most abundantly found in this case. The investigations are of more value in a negative way—we know that there were no fusiform bacilli of Vincent's, no Klebs-Loeffler or tuberculous bacilli. I was greatly disappointed with the laboratory investigation. The most impressive feature of this second case was the intense and penetrating odor of the necrotic tissue. It is also interesting to note in the two cases reported by me that the one represents the moist and the other the dry type of gangrene.

Unfortunately, no autopsy was attainable in either of the two cases which were under my observation. In the first case, for various personal reasons, it was not suggested; in the second I was, unfortunately, absent from the city at the time of the patient's death.

THE PREVENTIVE AND REMEDIAL TREATMENT OF ACUTE RHEUMATIC ENDOCARDITIS.[1]

By Beverley Robinson, M.D.,
of new york.

No practitioner of medicine who has had a dispensary or hospital service can help having an earnest purpose and wish to prevent, as far as possible, the development of acute rheumatic endocarditis. These feelings arise because of the keen appreciation, not of the great seriousness at the time of many cases of this disease, but of the woful and usually irremedial results which follow it. The larger number, as we well know, of all chronic cardiac disorders proceed directly therefrom, and when valvular disease is once established our power to cure organic lesions which are present is lacking, and even our ability to restrict the ultimate outcome of stenosis, insufficiency, or myocardial changes is often much limited. It is evident, therefore, that the study of prophylaxis in this affection is most important and far-reaching. We should effect prevention, not by directly combating the onset of endocarditis, but rather by

[1] Read by title before the Climatological Association, June 30, 1905.

aborting the attack of rheumatism which so often causes it. To-day, acquired knowledge points in many instances to the throat as being the door of entrance of the rheumatic poison. This statement is especially true of children. In diseased tonsils have been found streptococcus which, inoculated in animals, has produced articular rheumatism.[1] Further, Dr. F. A. Packard[2] reported five cases of endocarditis following acute angina. In three of these cases it was definitely known that the heart was normal before the attack of angina. During the attack endocardial murmurs developed; after the attack cardiac signs and symptoms persisted. This same author cites numerous instances reported by other observers, where cardiac lesions followed acute amygdalitis. Mention is also made of the frequency of cardiac lesions without the history of preceding rheumatism, but with the history of one or more attacks of severe sore throat. Sometimes the tonsillitis is present at the same time with other rheumatic manifestations, and it is difficult or impossible to affirm that the throat was the portal through which the rheumatic poison entered the system. Such a case, it seems to me, is one referred to by Dr. N. E. Brill,[3] where numerous cutaneous manifestations, together with involvement of several joints, followed the tonsillar inflammation. In this case a "blood culture was made and diplococcus tenuis was found, which, when guinea-pigs were inoculated, gave rise to arthritic pains and injured the valves."

To keep the throat in a very healthy condition is, therefore, a primary and essential indication. Not only is acute endocarditis thus prevented: if chronic endocarditis exists as a result of rheumatic infection, we can prevent in this way a renewal of endocardial inflammation. And even if sometimes we fail to prevent the development of tonsillar disease and the ensuing rheumatic attack, at least we may help the heart in a measure to withstand the invasion, and this in a sense is prophylaxis, according to Babcock. Tonsillar inflammation should be carefully and efficiently treated by local applications, and not only should acute manifestations be thus properly cared for, but all recurrent and chronic forms of nasopharyngeal, faucial and buccal disorder should be so governed that soon a normal condition of organ may be attained.

Whenever the use of sprays, gargles, and local pigments are unavailing, appropriate use of mild cauterizing agents is absolutely essential. Indeed, in some instances of enlarged, indurated tonsils, where the crypts are filled with foul-smelling, cheesy deposits, the microbic disease is not fully eradicated, short of operative procedure, followed by appropriate topical medication.

But general conditions must not be, and, indeed, cannot be,

[1] Article by Babcock in Medicine, March, 1904, p. 209.

[2] New York Medical Journal, April 29, 1905, p. 852; also Meeting of Association of American Physicians, May, 1899.

[3] New York Medical Record, November 12, 1904, p. 795.

ignored if we may expect the best results. The gastrointestinal tract is to be kept in an aseptic and fluent condition. All fermentations proceeding from careless, injudicious eating or dietary should be arrested with correctives or well-selected antiseptic agents. Satterthwaite[1] points out the correlation between intestinal disturbances and endocarditis, and speaks of instances where "in the course of an obstinate diarrhœa there was a sharp attack of endocarditis," and adds that "in the light of our present knowledge we may attribute it to gastrointestinal disorders."

But now comes an even more important condition to be considered and treated. This is the present and underlying rheumatic toxæmia. To prevent an outbreak, attention to diet, clothing, habits, surroundings, and occupation are very important. Good nutrition should be kept up by sufficient and suitable food. Wet and cold should be avoided. A suitable antirheumatic drug, or combination of drugs, should be taken immediately. And our choice seems at present to be confined to one of the salicyl groups, with or without alkalies or alkaline diuretics. Of the former, salicylic acid, one of its salts, and preferably the salicylate of soda, or ammonia, oil of wintergreen, or the synthetic methyl salicylate or salicin, the alkaloid from the willow bark, comprise the principal drugs which have received widespread commendation. Bicarbonate of potash or soda are the alkalies most employed; citrate, acetate, and nitrate of potash are the alkaline diuretics and refrigerants.

If the tonsillar inflammation is moderate and acute, small doses, three or four times in twenty-four hours, of some one of the antirheumatic remedies, is all that is required, and soon the beneficial results from their internal use is very obvious. The acute inflammation quickly subsides, and locally the mucous membrane again seems normal. If, however, the acute inflammation is simply grafted on a preceding condition in which chronic structural lesions are present, we shall in a few weeks or months have a recurrence of the disease, and it is only after more radical measures have been instituted that a permanent cure is obtained. In some of these instances the digestive organs already much disordered for a long while require particular attention in the manner already indicated. Whenever evidence of endocarditis, usually wholly objective and reduced simply to the expression of a systolic bruit at the apex, has been discovered and apparently developed without adequate cause, a neglected throat trouble has at times appeared to me clearly explanatory.

The first threatening outbreak of rheumatism may be that of the acute febrile disorder. So soon as the nature of the disease is defined antirheumatic medication should be immediately begun. The treatment at this time is most important. Not only should the right

[1] Diseases of the Heart and Aorta, New York, 1905, p. 42.

drug be selected, but it should be given at proper intervals and in sufficient dose. I have come to the ultimate conviction, partly by personal experience, partly by observation and analysis of my colleagues' hospital treatment, and in view, also, of my appreciation of what I have read, that no antirheumatic remedy is so useful and so free from objections as salicin. While I am not prepared by my own observation in acute rheumatism to endorse unqualifiedly the more than emphatic encomiums of Maclagan, I am inclined to believe from what I do know and have seen that he is substantially correct—viz., that salicin more nearly approaches a true antirheumatic specific, without great drawbacks, than any one we possess at present. Given in anything like a similar dose, salicylate of soda or oil of wintergreen will very soon occasion unpleasant or injurious effects. Oil of wintergreen causes head symptoms, in large or frequently repeated doses, which are annoying and frequently alarming. Salicylate of soda causes stomachal intolerance and, unquestionably, cardiac weakness, more or less enduring. Indeed, in cases in which it was probable that previous myocardial changes were present, the evil effects of its use were undoubted and lasting. Greenhow[1] states, for example, that "marked depression of the pulse and action of the heart" followed its use in almost every case—fifty in all. Further, he states it does not prevent or diminish "the frequency of complications in rheumatic fever." Against salicin no such objections can be urged, and even Greenhow, who, it seems to me, is somewhat too rigid in the proper estimate of its value, has not supported them. Speaking of salicin, Maclagan[2] writes: "It produces none of the deleterious effects of the salicylates. In several cases, some of which I have recorded, I have given full doses of salicin to patients suffering from the depressing and disturbing action of salicylate of soda; and under its use (though, of course, not in consequence of it) the depressing effects of the salicylate have disappeared." Salicin has been given in doses of 30 grains every hour for eight doses, and subsequently of 20 grains every two hours for one or several days, without occasioning appreciable accidents of any kind, and apparently with only relief to the patient's symptoms and general condition.

In the cases studied by Greenhow he gave the salicylate less frequently and in smaller doses, usually, than he did salicin. On the other hand, Preble[3] affirms that the salicylates "should be pushed to intoxication in the shortest possible time," and, further, that "the existence of a cardiac lesion is not a contradiction to their use." My personal observation would not support either of these statements. I would add, however, that David B. Lees insists also upon the great importance of giving large doses of the

[1] Clinical Society Transactions, 1886, vol. xiii. p. 244.
[2] Proceedings of the Medical Society of London, 1884, vol. vi. p. 112.
[3] Progressive Medicine, March, 1905, pp. 175-176.

salicylates in the rheumatism, acute and subacute, of adults and of children, but safeguards them with bicarbonate of soda. It is clear to my mind, however, that practitioners have usually erred in not giving larger and more frequent doses of salicin, especially in the initial stage of acute articular rheumatism, and so long as fever and pain in the joints were present. Of course, as Fagge[1] says, in speaking of the salicylates, the doses and frequency of giving which is essential to achieve good results, varies much with the case and the individual, and "it is generally necessary to use a larger quantity of salicin."

By the use of salicin the poison of rheumatism may be neutralized, the disease shortened, and thus there is less likelihood to have endocarditis appear. In those cases in which we do have endocarditis after treatment has begun, it may be that salicin was not made use of soon enough, and in sufficient doses. As treatment with it does no harm, it is wise in any event to persist in giving it, and, possibly, thus prevent further cardiac complications. It has been questioned, where salicin was given and no endocarditis developed, whether endocarditis would have occurred if no salicin had been given. There are no statistics to afford convincing proof, perhaps, and the favorable cases occasionally observed may have been accidental. And, after all, outbreaks of rheumatism vary at different times. It may be added, however, that "one[2] may reasonably expect that any remedy which has the power of arresting acute rheumatism, so that after its administration fresh joints no longer become affected, must also hinder the development of what is believed to be an analogous morbid process in or around the heart."

I believe salicin is such a remedy. Indeed, the report which I have from Dr. J. E. Traub, my former house physician at St. Luke's Hospital, New York, is to the effect that salicin has there proved most satisfactory of the salicyl group in the treatment of rheumatism, but as to its effect in preventing or curing acute rheumatic endocarditis, there was no evidence of a convincing kind to be obtained. Kinnicutt[3] believes that his cases treated with oleum gaultheria presented less than the usual proportion of heart complications. The late Dr. Flint,[4] Gottheil,[5] and Seelye[6] have reported analogous results. Greenhow[7] writes that salicylate of soda does not prevent or diminish "the frequency of complications in rheumatic fever."[8]

[1] Practice of Medicine, p. 557.

[2] Fagge. Practice of Medicine. Philadelphia, 1886, vol. ii. p. 537.

[3] Medical Record, vol. xxii. p. 505.

[4] Philadelphia Medical Times, vol. xiii. p. 846.

[5] Medical Record, vol. xxiv. p. 258.

[6] New York Medical Journal, November, 1884; National Dispensatory, 1894, p. 1125.

[7] Clinical Society Transactions, 1880, vol. xiii. p. 345.

[8] Methyl salicylate, or synthetic oil of wintergreen, now used in one New York Hospital by reason of false economy, is, I believe, an undesirable substitute for the natural oil. In many cases it is tolerated even less well by the stomach than salicylate of soda. According to latest information it may be that pure ethyl salicylate possesses several advantages over the natural as well as the artificial methyl salicylates. It is said to be less toxic than oil of wintergreen, and less irritating to the stomach than salicylate of soda (June 23, 1905).

In cases of recent valvulitis, Richard Caton[1] advocates strongly the use of iodides, "partly to help in the absorption of morbid exudations, but chiefly to lower muscular tension." He endeavors also to influence the cardiac vasomotor and trophic nerves reflexly, by gentle and almost painless stimulation of the first four dorsal nerves which we know to be in close relation with the heart. Babcock[2] is unwilling to accept any other evidence of the cure of acute endocarditis than what proceeds from autopsy findings, and his experience is not sufficient hitherto to enable him to be convinced of its possibility. "The mere disappearance of a cardiac murmur is not sufficient to justify a conviction in this regard." Clinical observation and pathology show, writes Babcock, that once acute endocarditis is thoroughly developed we have no means to cure it.

In instances of subacute and chronic rheumatism, where I have given salicin many times in small or moderate doses, more or less frequently, I have repeatedly known symptoms to disappear completely, or be much ameliorated in a few days or weeks, and without any intercurrent manifestations of an unfavorable nature which could be attributed to its use. I cannot say as much of the salicylate of soda, but shall make an exception for the ammonia salt which has stood me in good stead oftentimes, and particularly whenever there was marked stomachal irritability. We must ever bear in mind that while the heart may be attacked at the same time as the joints, we are not aware of it simply because it takes several days for the inflammation to be propagated from the fibrous to the serous layer, and then only through thickening and friction of the valves, occasion the condition which gives rise to an audible murmur—viz., minute or warty vegetations. The importance of this fact should be insisted upon both in the acute and subacute forms of rheumatism. Even when fully developed, Maclagan believes in the cure by means of salicin treatment and prolonged absolute rest of many cases of acute endocarditis. The importance of this rest cannot be insisted upon too strongly during the entire attack of rheumatic fever. Rest must be both mental and bodily. Thus the heart is freed additionally from sources of much increased action and tension, and it is evident in this way we lessen somewhat the chances of cardiac derangement.

In many of the ailments of children and adolescents, where they are closely watched, a marked rheumatic dyscrasia will be, if not at once, finally discovered. These affections differ materially with those of adult age. Articular symptoms are very infrequent; cardiac complications are the rule. Indeed, sometimes an endocardial murmur with fever is almost the first symptom of rheumatism

[1] Lancet, February 4, 1905, p. 301.

[2] Diseases of Heart and Arterial System, 1908, New York, Appleton & Co.

which is manifest. With it, however, may appear cutaneous symptoms, such as those which characterize erythema, purpura, recurring urticaria, or subcutaneous nodules. These nodules are occasionally seen over a superficial bone or tendon. Accompanying them there may be no fever and no articular manifestations. For Cheadle, the cutaneous disorder, accompanied by the endocardial bruit, is always a condition involving a very grave prognosis. If the rheumatic origin of any of these signs be recognized, we should save the heart from unnecessary strain and prevent, if possible, its becoming involved. Sometimes, despite rational precautions of prophylaxis, there may be an outbreak of acute articular rheumatism, and the heart may become affected. Indeed, this is so frequent as to seem to some observers a part of the normal march of the disease with children, and not a complication of it. It is most important to recognize it as soon as it does exist. This we may often do by a murmur or muffling of the first sound of the heart at the apex. There may also be an accentuation of the second pulmonic sound, or a reduplication of the second sound at the apex. (Parkinson.[1]) Irregularity and inequality of the pulse are deceptive, as they may occur in health. If our diagnosis of the presence of rheumatism be made, it should be promptly treated; first, by a proper amount of one of the salicyl group, and best of all by salicin; second, by absolute or relative rest of body and mind, depending upon the degree, stage, and locality of the rheumatic affection.

In giving the specific treatment of rheumatism, with salicin or a salicylate, care should be observed not to interrupt treatment too soon, and not to reduce the amount of the drug too rapidly. In either case, if we do, a relapse more or less serious of all previous symptoms is liable to occur and to last a longer or shorter time. What is true of one relapse is true in a very measured degree, unfortunately, of two or even more relapses. I am satisfied that with children especially, and when the heart becomes involved, as is usually the case, the salicylates cannot be persisted with in even moderate doses for a continued period, without doing considerable irremediable harm. In these instances I would insist most emphatically upon the proper use of salicin as being a far preferable, because a safer, remedy to administer. It may not act always so rapidly and at first, apparently, so usefully, but if our observations be careful and continued I am convinced all will agree with me and share my conviction. Despite treatment with salicin, however, these cases go frequently from bad to worse, and many of them result fatally, owing to an advanced condition of heart disease. While this statement is unfortunately true, I am confident more such cases are hurt unduly where salicylate of soda has been given to combat pain and lessen fever, by reason of its depressing action

[1] The Hospital, October 3, 1903, p. 10.

upon a heart with a myocardium already relaxed, softened, weak, and showing signs of beginning degeneration. In such cases it is wisdom frequently to diminish the amount of the salicylate and even of salicin, and combine with it a suitable amount of alkali. Dr. Gilbert Smith's tables, indeed, seem to show that the percentage of heart complication, at St. George's Hospital, London, "might be due to the fact that the practice of combining full doses of alkali with the salicylate was general" at that hospital. Indeed, the percentage was lower than at the other hospitals, and also than the general average of the presalicylate cases. This is in line somewhat with the statements of Fuller and Dickinson, who had shown that alkalies had a great influence in preventing heart complications.[1] The old alkaline treatment was essentially eliminant, and in my judgment, even if it does not shorten rheumatic attacks in adults or children, or prevent or cure endocarditis, usually, at least, it does no evident harm. Pursued moderately, during several days or weeks, and especially when we give therewith a suitable cardiac tonic or stimulant, such as sparteine, caffeine, or strophanthus, it relieves symptoms frequently, and without injurious after-results. David B. Lees[2] urgently advocates the necessity of combining with the salicylates at least double the quantity of bicarbonate of soda in order to prevent salicylate acid poisoning and the "air hunger" so characteristic of it.

For my own part, feeling as I do about the utility of biliary disinfection in the successful treatment of rheumatism, and recognizing the probable power in this direction of the salicylate group, and notably of salicylic acid, I am inclined at times to combine the use of chloride of ammonium with an alkaline diuretic like acetate of potash. I have assuredly seen more than once fever and pain lessened very notably by this combination, and in cases, perhaps, where the salicylates had been used already, without good results. The chloride of ammonium has been recognized for many a year as an hepatic stimulant and antineuralgic, among the most valuable. The late Dr. Anstie, of London, has interested me much, through his work on neuralgia, in the use of this salt. And a fact worthy of note is that the combination to which I refer was a favorite one with a down-town practitioner of New York, who, twenty to twenty-five years ago, had a very extensive reputation, even among the wealthy and cultured classes, for his great success in treatment of rheumatism in its subacute and chronic forms, of men and women, of middle life or beyond.

In acute exacerbations of the disease tincture of aconite was added with very great advantage to the relief of symptoms and comfort of the patient. The aconite was suppressed as the patient improved. In another toxæmia, namely, that of influenza, the

[1] Medical Society Transactions, vol. vi. p. 125.

[2] Acute Visceral Inflammations, London, 1904, p. 296.

alkaline treatment with chloride of ammonium is sometimes useful also, and particularly if there is any painful expression of the disease in connection with an endocardial murmur.

When any of the abnormal signs of rheumatism appear in a child, he or she should be confined to bed. And even though the child seems very little ailing, he must be kept there until all signs of rheumatic trouble have passed, either of heart or other organ. It is a small matter, writes Ashby,[1] to be overcareful in keeping in bed a child with slight joint trouble, but it is a grave affair to ignore acute endocardial trouble and to let children run about too soon. Usually endocarditis in children is accompanied also with myocarditis, and this is an additional reason for insisting upon rest in bed—viz., physiological rest. Indeed, there may be both pericarditis[2] and myocarditis, and thus all the heart structures are soon involved. Rest is even more important with children than with adults, as we know with them how liable the heart is to be attacked by the rheumatic poison, and how serious the remote results of it are. The task of absolute and prolonged rest in bed for children is a very difficult and ungracious one many times, but may occasionally be accomplished completely and efficiently, and ultimately with happy results. If the patient is made to understand in the beginning the great importance of this rest treatment, and how only in this way a complete cure can be expected, he or she, with wise parents, will submit to it. But it requires faith, courage, intelligence, will-power, and self-control on the part of physician, parent, and also, in a measure, of patient, to secure a thoroughly satisfactory result; I mean a complete cure. To no one more than to the late Dr. Maclagan, of London, and, latterly, to Dr. Caton, of Edinburgh, are we indebted for their wise insistence upon the all-important necessity of absolute rest in the treatment of acute endocarditis, at a time when it is yet possible to achieve a complete and permanent recovery. The guiding hand of the physician to prevent overstrain of the heart must be used not only during the period of absolute rest, but continued a long while afterward. This continued care and watchfulness is far more important in reality than the treatment of cardiac inadequacy due to permanent lesions when it has once developed. We perceive, then, that the physician's duty is not terminated when acute cardiac inflammation has disappeared. He must prevent chronic cardiac disease of valves and muscle and incompetency resulting therefrom, by wise counsel and avoidance for the patient of overfatigue. There is much for a careful physician to do in this direction, and he fails in duty if he does not instruct his patients who have had acute endocarditis what

[1] Ashby and Wright. Diseases of Children.

[2] In fatal cases, out of 150 only 9 escaped pericarditis, and at least one-fifth of the number had myocarditis. (Poynton.)

to do and what to avoid. Here, as Babcock writes, is "prophylaxis in the truest sense."

It is particularly important for patients affected with subacute rheumatism to take a complete rest for a while. In this way they will, I believe, often prevent the development of an attack of acute endocarditis. If a chronic valvular trouble already is present, this counsel of rest should be especially heeded, because if acute endocarditis becomes manifest it will surely aggravate the previous cardiac condition. It is wise to avoid all violent exercise during the period of convalescence. It is also prudent for the physician to see the patient frequently, so that the first distinct evidences of the presence of acute endocarditis may be immediately and effectively combated. Mental strain should be avoided as much as bodily effort. The former, like the latter, is apt to occasion cardiac inadequacy. This it does through augmented cardiac action caused by excitement, brain-fag, mental weariness. The slower the heart acts the longer the diastole, and hence the less violent its contractions. Unfortunately, the heart cannot be put at complete rest as the joints can, and for this reason it is very often difficult, or well-nigh impossible, to secure a complete cure. The heart should be quieted by the use of bromides internally. Thus the risk of rupture of the cusps through overaction or the transport of emboli to different organs is notably lessened. Digitalis should not be used to quiet the heart. While this drug slows heart and pulse, it increases the strength of cardiac systole and may occasion injury to the softened valves by the additional strain thus occasioned.

I confess I am somewhat skeptical in regard to the good effects of any local application in preventing endocarditis, unless combined with intelligent general treatment. In any event, as a preventive measure, I would not apply leeches, blisters, or even an ice-bag, with this object in view, unless there were rather pronounced symptoms indicating their use. The slight advantages possibly connected with their employment would be more than balanced by the annoyance which they would occasion. As regards the remedial effect of local applications, when acute endocarditis already exists, I hold somewhat different views. Whenever there is weakness and irregularity of pulse and heart, it is desirable to employ hot fomentations, and these, I am confident, are far better than cold compresses or an ice-bag, as their action is decidedly more stimulating. I am also satisfied that repeated fly-blisters over the precordial area are distinctly useful in a remedial way in commencing valvulitis, and, despite any nervous distress or pain occasioned by their employment, would insist upon them. Iodine, mustard, turpentine stupes, etc., do not take their place or act so beneficially.

In this connection I would cite James Tyson,[1] who believes "if

[1] Transactions of the Association of American Physicians, 1897, vol. xii. p. 208.

the blister is applied sufficiently soon, we may even avert the effusion" in pericarditis. Likewise, in endocarditis I believe we may avert roughening of valves and fibrinous deposit on them. Lees objects to the use of fly-blisters, on the ground that the soreness produced by them interferes with the physical examination of the heart. He admits, however, that they may be moderately useful in causing local muscular depletion. From this point of view he regards them as far inferior in value to the use of leeches. These, followed by the application, more or less continuously, of the ice-bag, diminish blood pressure in the right auricle, and when this portion of the heart is dilated, lessen its size and relieve symptoms very much, particularly dyspnœa and some lividity. An occasional mercurial is, in my judgment, advantageous. I do not believe aconite, veratrum viride, or tartar emetic are often indicated. In favor of the use of aconite I would say, however, that when the acute endocarditis is grafted on an old valvular lesion with general arterial changes and marked hypertension, it may exercise a valuable restraining influence in quieting the heart. It must be managed even then, however, with great care and good judgment, and not carried to the point where notable general depression followed its use.[1] What remains of treatment pertains mainly to allaying symptoms as they arise, and without having in view any evident and direct remedial effect. Thus pain and restlessness are combated with opium, or in children, preferably, with the bromides. Soothing liniments, locally, may also be advantageous. I rarely, if ever, make use of the coal-tar antipyretics, and, except phenacetin in very small doses, believe they invariably do harm. High temperature seldom exists or calls for hydrotherapy. The latter is indicated, as a rule, merely for the patient's cleanliness and comfort.

In the use of strychnine, where there is evidence of myocardial degeneration, I now rarely give doses beyond $\frac{1}{60}$ grain four times in twenty-four hours, as I believe, with Dr. Curtin and Dr. Jacobi,[2] the good effects are questionable with large amounts. Opium, in many such cases, when given in moderate doses, is a far preferable supporter of the heart. (Musser.[3])

In some of these cases of systolic bruit at the apex during acute or subacute, or even chronic rheumatism, we are in doubt for a time, shorter or longer, as the case may be, as to whether the cardiac murmur really proceeds from or is caused by the diseased endocardium, or is hæmic, or due to a dilated orifice caused, perhaps, by anæmia, by nervous derangement, or relaxed and softened valves, or cardiac muscle. Time, judicious treatment, prolonged rest, absolute or relative, will solve the diagnostic diffi-

[1] During the past winter I saw a case, in consultation, where aconite was undoubtedly useful, and particularly in affording relief to distressing cerebral symptoms.

[2] Discussion of Dr. Musser's paper.

[3] Paper read before the Association of American Physicians, Washington, May 16, 1905.

culties many times, and in any event are usually the best remedial means for a while, which we can rationally advise and, indeed, strenuously urge. With returning health both murmur and dilatation may disappear because the heart has regained its tone. Autopsies have shown that "one out of seven cases in which heart murmurs are heard no endocarditis is present In about one in seven cases in which endocarditis is found at autopsy no murmur was heard during life."[1] Again, as Jacobi[2] wisely states, "often we were apt to make a diagnosis of endocarditis when there was a myocarditis present." He had such cases under observation year after year; now and then they turned up with a systolic murmur (mitral). Then they would get better and remain so for months after treatment by rest, etc. The murmur would be brought out by the slightest exertion.

There is one remarkable and suggestive analogy in acute articular rheumatism, which has been insisted upon by more than one observer, although ignored, apparently, by many. It is the resemblance, in many particulars, to malaria. Even as regards the endocarditis we have many features which are striking and common, and in relapses of each disease the need of long continued, although at times intermittent, medication to achieve the best curative results.[3] Singular to say, the willow tree is most prolific in the soil and under ambient conditions where rheumatic toxæmia would most probably be furthered by secondary causes, just as the tree from which cinchona and its alkaloids are obtained is found in countries where malaria is endemic in most pernicious form, and just as coca comes from a region where the natives particularly require the staying power to ascend great mountain heights which mastication of the fresh leaves of the plant surely gives.

In rheumatism, in influenza, in cerebrospinal fever, and still other infections, similar symptoms often proceed, no doubt, from different microbes. It is possibly this latter element of the disease that explains, in some sense, at least, the alleged curative property of the diphtheria antitoxin in the late outbreak of acute meningitis.

"In view of the favorable results in some of the cases of mixed infection, one or more of the accidental toxins may have been neutralized, while the principal one that happened to be inconsiderable in virulency and possibly untouched was more easily overcome by a natural vital resistance of the patient. Such an assumption would give a reasonable explanation for the cases that proved fatal in spite of the new treatment."[4] It seems to me that this thought applies equally well to what we know to-day about

[1] Medical News, May 20, 1905, p. 958. [2] Medical Record, May 20, 1905, p. 1905.

[3] In an able paper, Dr. Warren Coleman has lately pointed out close resemblance also between malaria and infective endocarditis. (THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, March, 1905.) Query to my mind and to others is whether all cases of endocarditis are not infective. See Brill's case.

[4] New York Herald, April 7, 1905 (editorial).

the bacteriology of rheumatism and its frequent, at times almost essential, complication—acute endocarditis. This pathological condition occurs in a great many diseases, and it is a well-established fact that it follows many bacterial infections—syphilitic, pneumonic, streptococcic—after scarlet fever, rheumatism, and many others.[1]

Hence, I am of the opinion, and my clinical experience would seemingly corroborate it, that while salicin and the other members of the salicyl group relieve rheumatic symptoms in a remarkable manner in many instances, in other apparently similar cases they fail to do so. Why, we do not know as yet positively. In just such cases I feel confident that the rheumatic poison is still present and active; but we are unable to neutralize it, or in any event, with our ordinarily most effective remedy. In view, however, of clinical facts which show the similar pathological effects of different micro-organisms, we now endeavor to give an explanation which is rational and, provisionally at least, fairly satisfactory. Take one notable example to prove it. It is well known, and has been so for many years, that a Spa treatment at sulphur springs (Aix-les-Bains, Richfield, Sharon, etc.) has been judged to have positive therapeutic value in many cases of rheumatism, in which other remedies had ceased to be of value, or even, originally, had not responded favorably to our legitimate anticipations. Aside the theory of the causation of rheumatism, based essentially on the fact that it is due to the action of "diplococcus tenuis" or other form of micro-organism, it is evident that provisionally "we must be content to combat the symptoms of the disease, as they present, by purely empiric methods." (Conklin.)

In conclusion, I would emphasize again the importance of my subject for discussion—than which, indeed, I know of none of greater interest. It seems to me that to the late Dr. Maclagan and to Dr. Lees, both of London, the profession is under deep and lasting obligation for demonstration of facts fruitful of enduring advantage to innumerable patients.

NOTE.—During the past winter and spring I have suggested the use of salicin in the treatment of epidemic cerebrospinal meningitis. In several cases, at different hospitals of New York (St. Luke's, Bellevue, Gouverneur, etc.), salicin was given a trial, and, while it did not yield notable remedial results as regards the outcome of the disease, it did show marked power over the joint symptoms manifested in connection with it in a few instances. I am glad to note that apparently good results have been obtained latterly by Dr. A. Seibert[2] in cerebrospinal meningitis from rectal injections of large doses of sodium salicylate.

[1] Brill. New York Medical Record, November 12, 1904, p. 795.
[2] Medical Record, June 17, 1905. p. 930.

CHYLOUS AND CHYLIFORM EFFUSIONS INTO THE SEROUS SAC: WITH A CASE.[1]

By J. G. Wilson, M.D.,
of Philadelphia.

The subject of effusion of chyle, chyle-like and fatty fluids, has been especially investigated in this country by Busey, whose papers contain full studies of the early literature of the subject and important collections of cases. The paper read by Dr. Busey before this Society in 1889[2] contains a table of 33 collected cases of "effusion and accumulation of chyle and chyle-like, milky, fatty, and oily fluids in the peritoneal cavity," together with abstracts of 13 cases collated after the table was prepared. In Germany, Quincke (1875),[3] Bargebuhr (1893),[4] and again (1895),[5] Rotmann (1897),[6] Mutermilch (1902),[7] and others, have published valuable papers upon the subject.

To Quincke is due the credit of having first pointed out the distinction between chylous and chyliform effusions—hydrops chylosus and hydrops adiposus—while Bargebuhr recognized the important fact that a nice distinction between these two forms of effusion cannot in all cases be made, and that in some cases the presence of the chylous transudate and the chyliform exudate at the same time is by no means improbable—*gar nicht undenkbar sei.*

Bargebuhr collected 126 cases, including Busey's cases, reported between 1633 and 1894.

Rotmann's collection of 29 cases includes some that were overlooked by Bargebuhr and those published subsequently, making 155 in all to 1897. Of the latter 3 were personal observations made in Mosler's clinic. Since the date of Rotmann's paper many cases have been reported.

It is certain that the frequency of chylous and chyliform effusions is far greater than the literature indicates. The appearance of the fluid is striking, and at once arouses the attention of the trained observer; yet it presents in some cases a superficial resemblance to pus, which may mislead the physician whose attention has not been especially called to the subject. There are no means by which the nature of the effusion may be determined *intra vitam*

[1] Read by title at the Twentieth Annual Meeting of the Association of American Physicians, Washington, D. C., May 16, 1905.

[2] Transactions of the Association of American Physicians, vol. iv. p. 76; The American Journal of the Medical Sciences, vol. xc. p. 373.

[3] Deutsches Archiv f. klin. Med., Bd. xvi. pp. 121–139.

[4] Ibid., Bd. li. pp. 161–192.

[5] Ibid., Bd. liv. pp. 410–441.

[6] Zeitschrift f. klin. Med., Bd. xxxi. pp. 416–441.

[7] Ibid., Bd. xlvi. pp. 123–134.

except by the withdrawal of a portion of it. In many of the reported cases its presence was first recognized at the autopsy. The fact that cases occur much more frequently than the collected statistics indicate is evident upon the consideration of the experience of those who have written upon the subject. Senator saw 8 cases, Rotmann saw 5 cases at Greifswald in the course of two years, and I have seen in all 3 cases. Not all the cases by any means find their way into the literature, for the reason that many observers hesitate to report single cases and the cases which recover and those in which autopsy cannot be made are necessarily incomplete.

Rotmann's study of 155 cases shows the effusion to have been present in the abdomen in 104 cases, in the pleural cavity in 49 cases, and in the pericardium in 2 cases. In certain cases, as in that which forms the basis of this communication, the milky effusion has been found both above and below the diaphragm.

The pathological causes of milk-like effusion into the serous sacs are, in general: (1) trauma; (2) tuberculosis; (3) malignant disease; (4) filariasis; (5) thrombosis of the left subclavian vein; (6) disease of the liver; (7) syphilis.

The particular causes of such effusions into the peritoneal sac (*chylous ascites*) comprise those conditions which produce a solution of continuity in the vessel walls by injury, inflammation or extreme tension, or by transudation through the wall weakened by disease. Under this heading Rotmann has grouped the following special causes in the order of their frequency: Compression of the thoracic duct or the lymphatic vessels by glandular enlargements, neoplasms, etc., 17 cases; non-tuberculous peritonitis, 9; occlusion of the left subclavian vein, 6; excessive pressure, strain, cough, 5; peritoneal carcinoma, 5; filariasis, 3; occlusion of the thoracic duct, 3; occlusion in lymph glands, 3; occlusion of lymph vessels, 2; external pressure, 2; malignant lymphoma, 2; disease of the liver, 2; syphilis, 2; primary disease of the lymph vessels, angioma, 1; and calculus in the receptaculum chyli, 1.

The special causes of *chyliform effusions* into the peritoneal sac he groups as (*a*) due to fatty changes in cells, especially endothelial, and the cellular elements of new-growths—tuberculosis, 6; carcinoma, 5; cirrhosis of the liver, 3; cardiac disease, 2; sarcoma, 2; chronic non-tuberculous peritonitis, 1; and an apparently primary fatty degeneration of the endothelium, 1—and (*b*) an abnormal fatty condition of the blood—lipæmia, 1.

The special causes of *mixed forms* are, according to this observer, carcinoma, peritoneal, mesenteric, glandular, etc., 3, and tuberculous peritonitis, 1.

The special causes of *chylous effusion* into the pleural sacs are (*a*) those leading to the escape of chyle as the result of injury or disease of the vessels, namely, external violence, 8; carcinoma of the pleura, 5; occlusion of the left subclavian vein, 4; compression

of the duct by tumor, 2; malignant lymphoma, 2; disease of the lymphatic vessels, sclerosis, lymphangiectasis, 2; occlusion of the thoracic duct, 1; filariasis, 1; and excessive strain, 1; (*b*) the discharge of a chylous ascites into the pleural cavity by way of the lymph spaces, 1.

Rotmann groups the causes of *chyliform effusions* into the pleuræ as (*a*) the presence of cells that have undergone fatty degeneration, as in carcinoma of the pleura, 5; tuberculous pleurisy, 3; non-tuberculous exudative pleurisy, 3; abscess of the lung, 1; and (*b*) abnormal fat in the blood—lipæmia.

The two cases of fatty effusion into the pericardium in the series he attributes to rupture of chylous vessels (?) and the discharge of chyliform fluid from the left pleura by way of the anastomotic lymph channels. In 25 of the cases no satisfactory cause could be determined.

It is obvious from a review of the above facts that in a considerable proportion of the cases the causal conditions are such as to produce both chylous and chyliform products, and the conclusion is inevitable that both these factors must be present in many milky effusions, and that such fluids are often mixtures in varying proportions of transudates and exudates.

The chemical and morphological characters of milk-like effusions have been especially studied by Mutermilch. The investigations of this observer were conducted upon the fluid obtained by puncture in the case of a child aged five months, in whom a right-sided pleural effusion followed a fall from the mother's bed.

The quantity of the fluid withdrawn was 115 c.c. It presented the following characteristics:

A complete resemblance to milk; opaqueness; absence of odor; whitish color, with a faintly yellowish or creamy shade; feeble alkalinity; specific gravity, 1017. Upon microscopic examination there were seen great numbers of minute, dust-like granules in active, molecular movement. Here and there were slightly larger bodies, constituting distinct points scattered separately or in groups; a few leukocytes; larger cells containing distinct, highly refractive granules and erythrocytes in very small numbers, and only found upon careful search.

The chemical examination, conducted upon the hypothesis that the fluid was a fat emulsion, yielded the following results:

Shaken with ether in a test-tube the fluid became less opaque. When a few drops of potassium hydroxide were previously added it became quite transparent and almost colorless. Upon the addition of osmic acid the fluid became blackish.

Microscopically, upon the addition of a 1 per cent. solution of osmic acid to a drop of the fluid, the dust-like granules assumed a dark-gray color. The same change in color was observed in the granules in the large cells. Upon the addition of ether the

dust-like granules were dissolved. They ran together and formed at first smaller, then progressively larger, and finally quite large, highly refracting globules, which, upon treatment with osmic acid solution, became black in color.

The fluid, upon standing, deposited upon the bottom of the vessel grayish-red, flocculent masses, in which were to be seen under the microscope the above-described morphological elements.

The morphological elements were almost exclusively leukocytes and in marked preponderance lymphocytes. There were present in small numbers neutrophilic leukocytes containing fat granules, together with endothelial cells and a very few red blood corpuscles.

The chemical examination yielded the following results—100 c.c. of the fluid:

Solids	8.360
Albumin	6.135
Extractives by ether	1.481
Cholesterin	0.174
Soluble salts	0.563
Insoluble salts	0.028
Extractives by water	0.132
Extractives by alcohol	0.121

The fluid was absolutely sterile; neither microscopically nor by culture upon bouillon and agar were any micro-organisms to be recognized.

The above characters of the effusion in Mutermilch's case, considered in connection with the etiology of the condition—traumatism—and the entire absence of symptoms suggestive of pleural inflammation, justified the conclusion that the fluid consisted of chyle.

Quincke was the first to divide the fatty effusions into two groups: First, those consisting of chyle from the thoracic duct, or the lacteals—transudates—and, second, those resulting from fatty metamorphosis of the cellular elements in inflammatory effusions—exudates. It was generally thought that these two forms of milky fluid could be differentiated only by the microscope. Quincke described a case in which there was escape of chyle into the pleural cavity. The fluid was like milk, with a slightly yellow shade. Under the microscope it showed minute granules of different size, but all in the form of apparently round points, or dust particles, and a very small number of white blood corpuscles.

In contrast to this chylous effusion he described the form of fatty fluid resulting from fatty changes in the morphological elements. The fluid was bright yellow, somewhat cloudy, whey-like. Upon standing there formed upon the surface a thin, cream-like layer, showing under the microscope small globules, mostly in the form of collections of highly refractive granules, less frequently having sharp borders, and still less frequently presenting the appearance of large, indistinct nuclei. In the fluid underlying this layer were

seen white blood corpuscles and larger round cells, which, in consequence of differences in the amount of fat, presented all possible transition forms. In this case Quincke ascribed the presence of the fat to the breaking down of cellular elements. He designated the effusions of the first category as hydrops chylosus; those of the second category as hydrops adiposus.

In 1885 Senator[1] adopted Quincke's division of the fatty effusions into hydrops chylosus and hydrops adiposus. Since the cases in the second category result from the fatty metamorphosis of endothelial and other cellular elements, and the diagnosis rests upon the presence of cells in which the fatty changes are incomplete, it is obvious that in advanced cases, in which the transitional forms may not be found, the diagnosis of the nature of the effusion must remain obscure. For this reason Senator ascribed great importance to the chemical study of these fluids, and especially to the presence of grape sugar, holding that chyle and lymph contain sugar constantly in quantities which may be easily recognized, while this substance is only found in serous transudates and exudates in non-diabetics exceptionally and in traces. Grape sugar, in more than doubtful traces, was believed by this author to indicate the presence of chyle. Later investigations, especially those of Pascheles and Reichel,[2] Pickardt,[3] and Rotmann,[4] have established the fact that ordinary serous transudates and exudates often contain this substance in recognizable quantities, and that its presence in chyliform exudates may also be expected.

Strauss[4] was able to demonstrate the chylous nature of the fluid in fatty ascites by giving his patients a diet rich in fat, the administration of which was found to greatly increase the proportion of the fat in the peritoneal fluid. This observer regarded the rapid renewal of the fatty fluid having always the same peculiarities as indicating its chylous nature.

The milky effusions into the serous cavities do not always owe their characteristic appearance to fat. Micheli and Mattirolo[5] have described pseudochylous forms of ascites, and have satisfied themselves experimentally that in the cases which they had under observation the milky appearance arose from the presence of lecithin.

Dr. Edsall reported at the last meeting of the American Pediatric Society a case of Hodgkin's disease, not yet published, in which there was a non-fatty pleural effusion, the opacity being due to altered globulins.

The case about to be described, brought to me by Dr. Housekeeper, of Vandengrift, Pennsylvania, illustrates one of the more

[1] Charité-Annalen, 1885, p. 307.
[2] Wien. klin Wochenschr., 1896, No. 17.
[3] Berl. klin. Wochenschr., 1897, p. 844.
[4] Münch. med. Wochenschr., 1898, No. 6.
[5] Archives de physiol. norm. et patholog., 1886, p. 367.
[6] Wien. klin. Wochenschr., 1900, No. 8. p. 56.

common forms of fatty effusion into the serous cavities. It may, I believe, be properly regarded as an example of that group of cases in which the effused fluids, though fatty, are neither chylous nor chyliform or chyloid, but a mixture of both. This opinion is based upon the following facts:

1. The constant gross physical characters of the peritoneal fluid and that removed from the left pleural cavity upon repeated aspiration on several occasions at comparatively short intervals of time.

2. The practically identical microscopic and chemical characters of the two specimens of left pleural effusion examined by Dr. Kelly and obtained within a period of twelve days.

3. The presence in those fluids of fine dust-like granules in great numbers, and showing active molecular movement and lymphocytes, together with polynuclear neutrophiles, nuclei, and endothelial cells in various transitional stages of fatty change.

4. The different gross and microscopic characters of the fluid contained in the circumscribed right pleural effusion.

5. The underlying pathological conditions, namely: diffuse sarcoma of the retroperitoneal and thoracic lymph glands and inflammatory pleural changes.

6. The softened condition of the lymph glands in the neighborhood of the receptaculum chyli, and the fact that the walls of that structure could not be recognized upon careful search and the thinned walls of the thoracic duct, changes permitting the escape and transudation of chyle.

7. The inflammatory changes about the sarcomatous glands and the pleural inflammation, the source of proliferation of endothelial cells and serofibrinous and seropurulent exudate, with fatty changes in its cellular elements.

Sarcoma of retroperitoneal and thoracic lymph glands; fatty ascites discovered upon operation for hernia; double pleural effusion, fatty upon the left side and presenting upon repeated aspiration, at intervals extending over a period of many months, the same appearance; circumscribed and seropurulent upon the right side; microscopic and chemical notes on the fluids; autopsy.

Anamnesis.—A. M. B., female, aged thirty-five years, single, a teacher; admitted to private room in the German Hospital December 5, 1904.

Family history good; father living and well; mother died about the menopause from disease the nature of which is unknown to patient; only two brothers, who are living and in good health; no sister. No history of tuberculosis or malignant disease could be obtained.

The patient states that she had scarlet fever in childhood, but cannot tell whether or not she had the other diseases of childhood. She had a severe attack of tonsillitis at the age of fifteen, but always

regarded herself as having good health until about three years ago. Until that period she had a good appetite and digestion and regular bowels. First menstruated at fourteen, and that function has been regularly and painlessly performed until the present time. Dates her illness from the appearance of a femoral hernia upon the right side in the spring of 1902. This speedily began to give trouble, which led to an operation shortly afterward. In reply to a note of inquiry which I wrote to her surgeon, Dr. J. J. Buchanan, of Pittsburg, I received the following reply: "I operated on Miss A. M. B. for the radical cure of a femoral hernia with irreducible omental contents, if I remember aright. The peritoneum contained a considerable quantity of free fluid which was opaque, milky, or deep straw color. I remember that she was turned on her side to allow it to flow out. Perhaps there was a pint or two. No tubercles were on the sac or presenting omentum. She had no fever before or after the operation. Her wound healed *per primam.*"

The fluid was thought to be that of a tuberculous peritonitis. I was unable to ascertain whether or not a systematic examination of it had been made.

The following autumn she began to be short of breath upon exertion and had during the winter four severe attacks of bronchitis. In July, 1903, she had another attack of bronchitis. In the intervals between these attacks she had occasional slight cough and little or no expectoration, but felt constantly oppressed and was very short of breath upon even moderate exertion. At the close of the last attack of bronchitis the presence of a left pleural effusion was recognized and paracentesis was performed and a quantity of fluid, milk-like in appearance, withdrawn. This operation has been repeated a number of times, in all, as follows:

1903. July	quantity withdrawn about	.	.	.	1000 c.c.
1904. July	" " "	.	.	.	1000 "
1904. October 1	" " "	.	.	.	2000 "
1904. October 15	" " "	.	.	.	2550 "
1904. November 1	" " "	.	.	.	2500 "
1904. December 2	" " "	.	.	.	2500 "

The fluid always presented the same appearance and corresponded, according to the patient, to that which was removed after she came under my observation, and is fully described in Dr. Kelly's report which follows. During this period the patient occasionally noticed a slight discharge of similar fluid from the scar in her thigh resulting from the operation for the radical cure of the hernia. Her urine has never been milky in appearance. About the time that the first aspiration of the chest was performed her cough ceased and has not recurred. She has had no chills, fever, night-sweats, headache, or pain. Two years ago she weighed 128 pounds. Her present weight is 108 pounds.

Status Præsens. A tall, slender, well-formed woman of good color, but slightly cyanotic on exertion. No dropsy or œdema; no

cutaneous eruption. Temperature normal, at times slightly subnormal; chest somewhat flattened on both sides, but more upon the left than upon the right. Respiratory movement shallow, the excursus being much more limited upon the left than upon the right side. Respiration frequency while at rest 20, upon slight exertion rapidly increased to 30 to 36.

The impulse of the heart not well made out either upon inspection or palpation. It is interesting to note in view of the postmortem findings that careful palpation and auscultation failed to reveal any signs of a mitral stenosis. In the precordial area there was no thrill, nor was any murmur to be heard. There is accentuation of the second sound at the base, particularly in the pulmonary area. The pulse is small and of moderate tension. Its frequency varies in repose from 80 to 96.

The percussion resonance is clear on both sides anteriorly in the infraclavicular regions, with a slight tympanitic quality upon the left side. Below this it is impaired upon the right side and there is dulness below, with flatness at the base upon the left side. The percussion signs posteriorly correspond to those in front except that dulness (flatness) extends much higher and there is flat tympany at the upper part of the chest. There is partial obliteration of Traube's semilunar space.

Upon auscultation feeble vesicular and bronchovesicular respiration over the upper lobes, but no rales. At the right base posteriorly a few faint subcrepitant and crepitant rales, especially upon deep inspiration. Over the area of dulness on the left side absent breath sounds diminished vocal resonance. There is absence of vocal fremitus. No friction sounds except in a limited area at inner border of left scapula. The area of liver dulness not enlarged. Its lower border corresponds to the margin of the ribs in the midclavicular line and descends about two fingers' breadth upon full inspiration.

The lower border of splenic dulness is below the costal margin.

The abdomen is soft, elastic, and symmetrical. The presence of ascites cannot be made out.

Urine amber, acid, 1030; no albumin; no sugar; phosphates, epithelial cells, leukocytes, and bacteria. Amount measured 400 to 900 c.c. Some lost at stool.

Blood: hæmoglobin, 90 per cent.; erythrocytes, 6,325,000; leukocytes, 7200.

Repeated examination both by day and by night showed no filaria.

Operation December 19, 1904, by Dr. Deaver. A large portion of the thoracic fluid was removed with aspirator. An incision 12 cm. long was made parallel to and over the eighth rib, its median portion corresponding to the midscapular line. Rib exposed and intercostal vessels raised upward; a bone elevator placed under rib

and about 5 cm. removed with lion-jawed forceps. A similar portion was excised from rib above. Intercostal vessels ligated. Thoracic cavity opened, a quantity of chyloid fluid evacuated with force. Not all the fluid was removed. A large rubber tube (short) was anchored in the incision with silk. Incision was closed up to the tube with catgut. Antiseptic dressing.

For a time after the operation the patient did well, the temperature remaining normal. Beginning upon the tenth day the temperature became irregular, at times reaching 102° or 103°, and falling again to normal. The patient became dyspnœic and somewhat cyanotic with rapid pulse, which upon January 1st became uncountable. Upon January 3d the temperature rose to 103°, and the patient gradually sank, dying at 5.30 A.M.

The following is Dr. Kelly's report of his examination of the fluid removed from the left pleura December 8, 1904:

The fluid quite resembles milk in ordinary appearance—being whitish or slightly yellowish, opaque, and odorless. Its specific gravity is 1017. It contains a considerable amount of albumin—roughly, about 4 per cent. It does not respond to the ordinary tests for sugar—copper solutions, phenylhydrazin, and fermentation tests. The addition of ether followed by vigorous agitation causes the fluid to clear somewhat; the previous addition of a few drops of potassium hydroxide causes it to become much clearer. The addition of osmic acid causes the fluid to become quite blackish. On standing sometime a slight fibrinous deposit settled at the bottom of the vessel, and a small layer resembling the cream that forms upon milk formed at the surface. After standing in stoppered test-tubes and flasks at room temperature for ten days and two weeks, and being in the mean time several times exposed to the air, the fluid appeared quite "sweet."

Microscopically the fluid consists of cells (the character of which will be discussed presently) and a very large number of the finest granules; occasionally a single or several larger granules are observed, but almost all are very fine, even dust-like. They exhibit considerable molecular movement. The addition of a few drops of 1 per cent. osmic acid solution to the microscopic preparation causes the free granules, as well as similar granules in the under-described cells, to become darkish, blackish-gray in color. The addition of ether results in solution of the granules and their confluence to form larger masses, which become discolored, blackened, on the addition of osmic acid.

In addition to these granules, the fluid contains a moderate number of cells, a differential count of which reveals the following: lymphocytes, 76 per cent.; large mononuclear cells, 22 per cent.; polynuclear neutrophiles, 2 per cent.

The lymphocytes conform in all particulars to the lymphocytes of the circulating blood, and the polynuclear neutrophiles are also

readily recognized as identical with the predominating leukocyte of normal blood. The cells described as large mononuclear differ somewhat in appearance among themselves. A few of them resemble the lymphocytes, though they are larger; others recall the large mononuclear leukocyte of normal blood; but on account of the presence on the cells of granules resembling and behaving toward ether and osmic acid like the free granules of the fluid, of vacuolation, and of degenerative changes, it is impossible to say what some of these cells are. Other cells, however, are roundish in outline, vary somewhat in size, have a fairly large, usually eccentric nucleus; their protoplasm contains fatty granules and vacuoles; evidently they are desquamated endothelial cells.

Appropriate staining methods failed to reveal any bacteria in cover-slip preparations.

By culture methods a large unidentified, spore-bearing bacillus (probably contamination) was isolated.

The results of the examination of a second specimen of fluid received December 20th was identical with the foregoing (except that one portion was slightly pinkish, owing to the admixture of blood).

Almost daily examination of the blood (by day and by night) between December 8th and 20th failed to reveal filaria sanguinis hominis.

A specimen of the fluid was handed to Dr. Charles A. Fife, of the William Pepper Laboratory of Clinical Medicine, for chemical examination. He reports as follows: The fluid is faintly alkaline to litmus paper, but acid to phenolphthalein, requiring 20 c.c. of a one-tenth normal sodium hydroxide solution to neutralize 100 c.c. of the fluid. The specific gravity is 1016.4.[1] The fluid consists of 93.388 per cent. water and 6.61199 per cent. solids, of which 4.679 per cent. is albuminous (probably entirely serum albumin), 1.065 per cent. fats, and 0.5639 per cent. ash. The fluid after the removal of the fats and proteids reduces copper slightly, but does not respond to the phenylhydrazin sugar tests.

The following are the notes of the necropsy (No. 735), made by Dr. A. O. J. Kelly, January 3, 1905, six hours after death:

The body is that of a fairly well developed female subject with gracile bony development, slight musculature, and slight panniculus adiposus. The skin is pale; the nutrition poor. There is slight postmortem rigidity, but no postmortem lividity. In the right groin there is a linear scar (the result of an operation for femoral hernia). Posteriorly in the left thorax there is an opening into the pleura, a defect of a rib (due to resection), and a rubber tube draining the pleura. The preperitoneal adipose tissue is slight in amount. The thoracic and the abdominal viscera have their normal relations the

[1] Determined by a more sensitive instrument than was used in the other examination.

one to the other. The left pleura is non-adherent, except for a small area on the diaphragmatic surface; it contains 200 c.c. of rather thick "milky" fluid; in general it is pale, cloudy, and thickened, being in some places from 2 to 3 mm. thick; and it is covered (both the parietal and the visceral layers) with a rather thick or viscid, whitish, opaque, "milky" exudate. The left lung is small and compressed, and weighs 400 gm. It is mottled, grayish-black in color, of increased consistency, contains less air than normally, and reveals slight hypostatic congestion. The section surface is smooth, moist, and glistening, pale grayish in color, and exudes considerable frothy fluid. The lung crepitates throughout, and contains no tubercles and no cavities. The bronchi contain some serous or mucous fluid; their mucous membrane is pale, their calibre normal, but about them and extending somewhat into the adjacent tissue, there is apparently a slight amount of peribronchial connective-tissue overgrowth. The right pleura is dull, cloudy, pale, thickened, and universally adherent. As far down as the fourth rib the adhesions are firm and quite obliterate the pleural cavity. Below the fourth rib the adhesions are not so firm, and instead of obliterating the pleural sac, give rise to the formation of sacculi containing fluid; between the inferior surface of the lung and the diaphragm there is a large collection of fluid displacing the lung upward, but the lung extends down dome-like over the fluid—its edges reaching well down anteriorly, laterally, and posteriorly. The pleura contains a total of 550 c.c. of fluid, which, although somewhat "milky," is more correctly dull grayish in color, opaque, and "thinner" than the fluid obtained from the left side. The right lung weighs 650 gm. It is mottled grayish-black in color, of increased consistency, contains less air than normally, and reveals slight hypostatic congestion. The section surface is smooth, moist, and glistening, pale grayish in color, and exudes considerable frothy fluid. The lung crepitates through, and contains no tubercles and no cavities. The bronchi contain some serous or mucous fluid; their mucous membrane is pale, their calibre normal, but about them and extending somewhat into the adjacent tissue there is apparently a slight amount of peribronchial connective-tissue overgrowth. A few of the peribronchial lymphatic glands are quite small, firm, and anthracotic, and reveal no deviations from the normal; the great majority of them, however, are much enlarged, some of them very much enlarged, pinkish in color, and softened—almost diffluent. The pericardium contains 30 c.c. of clear, amber-colored fluid, and is non-adherent throughout. The heart is dilated, somewhat rounded in shape, and weighs 230 gm. It, more especially the right side, contains considerable dark clotted blood. The left auricle, the right ventricle, and the right auricle are dilated, especially the right auricle. The mitral orifice is contracted, and the leaflets thickened, indurated, and shortened. The chordæ tendinæ are also thickened and shortened.

The tricuspid orifice admits three fingers readily, and the valve leaflets are probably insufficient. The aortic and the pulmonary valves are normal. The left ventricle measures 8 to 13 mm. in thickness, the left auricle 2 to 4 mm., the right ventricle 5 to 8 mm., and the right auricle 1 to 2 mm. The heart is pale, brownish-red in color, and firm in consistency. The coronary arteries are normal. The arch of the aorta and the descending aorta are slightly thickened in patches. The œsophagus is normal. The omentum is normal in size and slightly fatty. The peritoneum is dull, cloudy, pale, and slightly thickened in places, especially the peritoneum of the anterior abdominal wall and that of the posterior peritoneal cavity toward the vertebræ near and extending into the pelvis. It is non-adherent throughout. It contains 1200 c.c. of grayish-white, opaque, "thin" fluid. The liver measure 27 x 18 x 5.5 cm., and weighs 1340 gm. It is normal in shape, of increased consistency, smooth on the surface, and has sharp edges. The section surface is smooth, glistening, and moist, mottled yellowish-brown (nutmeg) in color, and exudes an increased amount of blood. The gall-bladder is normal in size, contains fluid bile; the cystic, the hepatic, and the common bile-ducts are patulous. The spleen measures 11.5 x 6 x 2.5 cm., and weighs 90 gm. It is dark in color; the section surface is smooth; and the trabeculæ are slightly increased. The right kidney measures 10 x 5.5 x 3 cm., and weighs 130 gm. The capsule is slightly adherent, and when stripped leaves a slightly granular surface, dark in color. The kidney in general is of increased consistency. The cortex is 7 mm. in thickness. The left kidney measures 11 x 5.5 x 3 cm., and weighs 150 gm. It is similar to its fellow in appearance. Both ureters and both adrenals are normal. The urinary bladder and the external and the internal genitalia exhibit no deviations from the normal aside from the ovaries, which are slightly enlarged, whitish, firm, and cystic. The stomach is normal in size and position; its mucous membrane is slightly congested, especially on the posterior surface (postmortem). The entire intestinal tract appears normal. The pancreas is pale pinkish in color, quite firm, and exhibits no noteworthy deviations from the normal. The mesenteric glands are slightly enlarged, pinkish in color, and somewhat softened. The retroperitoneal glands, especially those just above the promontory of the sacrum, are much enlarged, quite red in color, and very soft, and many of them being quite diffluent; indeed, such is the softening and diffluence that it is impossible to distinguish gland from gland; higher up glands may be differentiated, but all are enlarged, softened, and red or pinkish in color. About the normal situation of the receptaculum chyli there are several enlarged glands scarcely to be differentiated from adjacent structures, but no receptaculum as a separate structure can be made out. Dissecting out the thoracic duct as high up as the clavicle no noteworthy change can be recognized—in particular

it is not enlarged or thickened. The brain and the spinal cord are not examined.

Anatomical Diagnosis. Sarcoma of the mesenteric, retroperitoneal, and peribronchial glands; bilateral pleuritis with chyliform exudation; chyliform ascites; mitral stenosis and insufficiency; dilatation of the heart; slight sclerosis of the aorta; passive congestion, œdema, and cyanotic induration of the lungs; passive congestion of the liver, spleen, and kidneys.

The following is the result of the microscopic examinations:

The enlarged mesenteric, retroperitoneal, and peribronchial glands are the seat of a new-growth that has almost entirely replaced or destroyed the lymphadenoid tissue—to a varying degree, however, in different glands. The new-growth consists of elongated, for the most part, spindle-shaped cells, with ovoid, rather vesicular nuclei, that stain well. The cells are arranged parallel or almost parallel the one to the other, but they are collected in fasciculi that course in varying directions, whence longitudinal, transverse, and oblique sections of the fasciculi are encountered. In many places the fasciculi seem limited to the sinuses, being apparently surrounded by a layer of flat endothelium; but in many places such arrangement and limitation are altogether wanting, and there is no appearance that warrants one in stating positively that the cells have been derived from proliferating endothelium. But little or no intercellular substance is apparent (by the ordinary methods of staining). Bloodvessels are comparatively few; those present have thin walls, mostly endothelium only, and they course amidst encircling fasciculi of tumor cells. Many of the retroperitoneal glands, especially those near the promontory of the sacrum, exhibit moderate necrosis. The other glands exhibit no retrograde alteration.

The pleura of the left lung is markedly thickened, the thickening involving the subendothelial connective tissue, the endothelium itself being wanting. This thickening consists of a highly vascularized fibrocellular tissue, the proportion of cells and fibrillar intercellular tissue varying in different regions. In general toward the free edge of the pleura the cellular collection consists of young fibroblasts and the ordinary round cells of inflammation; and the changes here merge rather rapidly into the more ordinary changes of fibrinous and fibrinopurulent exudation—a thick layer of which covers the pleura. The pleura of the right lung exhibits the usual lesions of adhesive pleuritis with considerable round-cell infiltration. Throughout both lungs there is an unusual thickening of the alveolar walls, which, however, is not uniformly distributed; in regions where it is less marked, there is considerable distention of the intra-alveolar capillaries, but where the thickening is more marked the capillary distention is obscured or absent. The thickening consists of a fibrocellular tissue rich in cells with elongated nuclei that stain well. Most of it appears to be white fibrous tissue, but

there is a considerable proportion of elastic tissue. In some places the cellular new-growth is excessive and completely obliterates the lumen of several adjacent alveoli. One receives the impression that here and there there is an unusual proliferation from one side of the wall of the alveolus into its lumen; in other regions, doubtless because of transverse section of such a polypoid growth, the new tissue reposes in or fills an alveolus without manifesting attachment to its wall. In addition to this connective-tissue overgrowth, many of the alveoli are more or less filled with an exudation consisting of leukocytes, desquamated epithelium, and a few erythrocytes. Many of the desquamated epithelial cells are excessively pigmented. This alveolar exudation is most marked just below the pleura of the left lung, where, together with the proliferation of the connective tissue of the alveolar walls, it gives rise to almost complete obliteration of the lumen of many alveoli.

The liver exhibits moderate dilatation of the central veins and adjacent capillaries, moderate pigmentation, and slight atrophy of the regional liver cells. The spleen exhibits considerable thickening of the trabeculæ, and considerable pigmentation—partly free and partly within the cells of the pulp. The kidneys exhibit moderate passive congestion and slight degeneration of the epithelium of the convoluted tubules. The uterus exhibits a slight grade of hyperplastic glandular endometritis. Otherwise there are no noteworthy microscopic alterations in the organs.

The fluid from the right pleural sac has a specific gravity of 1017. It contains 4 per cent. of albumin (Esbach). Microscopically there is considerable fibrin and a moderate number of cells, but none, or very few, of the fine granules observed in the fluid obtained by puncture from the left pleura during the life of the patient. A differential count of the cells reveals the following: lymphocytes, 58 per cent.; large mononuclear cells, 25 per cent.; polynuclear neutrophiles, 17 per cent. The lymphocytes and the polynuclear neutrophiles are readily identified. Some of the large mononuclear cells are doubtless degenerated lymphocytes, but the majority appear to be more or less degenerated and vacuolated endothelial cells.

The fluid obtained from the peritoneum has a specific gravity of 1016. It contains 3.5 per cent. of albumin (Esbach). Microscopically there is a moderate number of cells, and a large number of granules identical with those obtained from the left pleura. The granules are free and also contained within certain of the cells. They stain blackish with osmic acid, and are dissolved with ether. A differential count of the cells reveals the following: lymphocytes, 70 per cent.; large mononuclear cells, 29 per cent.; polynuclear neutrophiles, 1 per cent. The large mononuclear cells are similar to those found in the fluid from the left pleura, though they differ among themselves. Many of them contain fine granules that are

dissolved by ether, and stain black with osmic acid. They are doubtless desquamated endothelial cells.

Cultures from the right pleura and from the left pleura revealed staphylococcus pyogenes albus and aureus. Cultures from the peritoneal fluid revealed bacterium coli communis.

Within five hours of making the necropsy I inoculated three rabbits subcutaneously (in the William Pepper Laboratory of Clinical Medicine) with an emulsion of the softened retroperitoneal glands. One rabbit died the following day from peritonitis due to puncture of the intestine with the inoculating needle. The other two rabbits lived in excellent health, and were finally killed on April 6, 1905. No lesions were found at the necropsy.

REPORT OF A CASE OF METASTATIC ENDOTHELIOMA.

By Edward Jenner Wood, S.B., M.D.,
of Wilmington, N. C.

The patient was a white man, aged forty-three years. His occupation was that of a salesman. There was nothing in his family history of any note except that his father died of carcinoma of the œsophagus, and a brother died of a complication of cardiac and renal affections. There was nothing in his previous medical or surgical history of any importance until the year 1900. During that year he noticed a small nodular mass on the surface of the right testicle. The testicle became painful and began to increase in size. It was firm in consistence and showed no sign of breaking down in any part of it. This condition gradually increased until the final removal of the testicle. In the opinion of the surgeon the growth was sarcomatous.

After a lapse of over two years, in 1902, the patient felt the presence in the remaining testicle of the same symptoms that formerly had attracted his attention—namely, the nodule of the size of a buckshot, the increase in consistence, the swelling, and the pain. Again he was advised to have the radical operation performed, which he consented to. In this instance the histological examination was made by a competent pathologist, who pronounced it a large round-cell sarcoma.

Following this second operation the patient's health improved, though he was never robust. In September, 1904, following a fall, he began to complain of backache. Seldom was he free from pain, which usually was of the dull, boring character, though at times it was of the stabbing variety. The patient thought that this more severe pain was relieved coincidentally with the voiding of his urine.

This pain persisted with intervals of comparative freedom followed by exacerbations which would necessitate his remaining in bed several days at a time. In many respects this pain simulated lumbago. During this time the patient kept his full weight and showed no signs of cachexia.

In January, 1905, he came under my care with the simple history of pain in the lumbar region. This pain was most obstinate and resisted the largest doses of morphine. About the middle of January I detected in the left upper quadrant of the abdomen, just within and below the splenic area, a globular-shaped mass which was absolutely immovable in any position the patient might assume. This mass was uncovered by gut and was perfectly flat on percussion. The spleen was normal in size and position. The left kidney could not be palpated in its normal position, while the right was palpable. All other viscera were normal to physical examination.

The provisional diagnosis of malignant disease of the left kidney was made and it was advised that an exploratory incision be made with a view to a possible nephrectomy. The exploratory incision was made and revealed directly beneath the peritoneum a kidney enlarged to about three times the normal size, being irregular in contour and consistence, which was placed almost transversely across the abdomen, corresponding to a line drawn from the gall-bladder to the spleen. The organ was bound down to the lumbar spine by very dense adhesions, and could not be moved a fraction of an inch. It transmitted the pulsations of the abdominal aorta. The surgeons found a nephrectomy to be impracticable and the wound was closed with nothing done. He rallied feebly from the shock of even this slight operation.

A few days later it was noticed that the right leg was considerably œdematous and there was a marked difference in the temperature between it and the left leg, which was much warmer. It gave the patient considerable discomfort, though not acutely painful. The œdema soon extended to the scrotum.

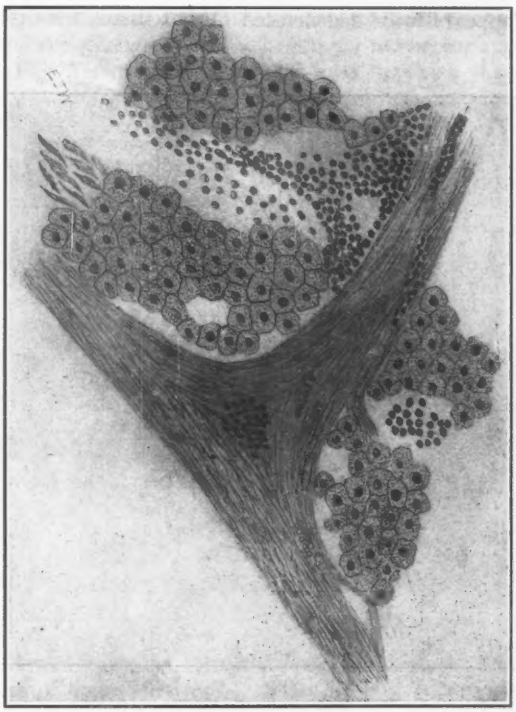
For a few days the patient was free from the lumbar pain, but it soon returned with added violence and could not be relieved by morphine.

Ten days after the operation the patient was seized with pain in the side, over the base of the right lung and became cyanosed. For a short time his condition was grave, but he rallied. Just previous to this he had begun a "prune-juice" expectoration, which now became very fetid and was expelled by a painful cut-off pleuritic cough. The temperature, which hitherto had been normal, now rose to 104°, and delirium developed. It was apparent that the condition was gangrene of the lung. Twenty-four hours later the patient died with a large pulmonary hemorrhage.

An autopsy was immediately done. The tumor mass was found to be the left kidney. It was bound to the spine by such dense

adhesions that it could only be torn out piecemeal. There was nothing that resembled normal kidney tissue and, in fact, it was structureless save in small areas, the bulk being made up of a mass of soft caseous material. At one point it was broken down. The right kidney was normal excepting a slight increase in size which was probably compensatory hypertrophy.

FIG. 1



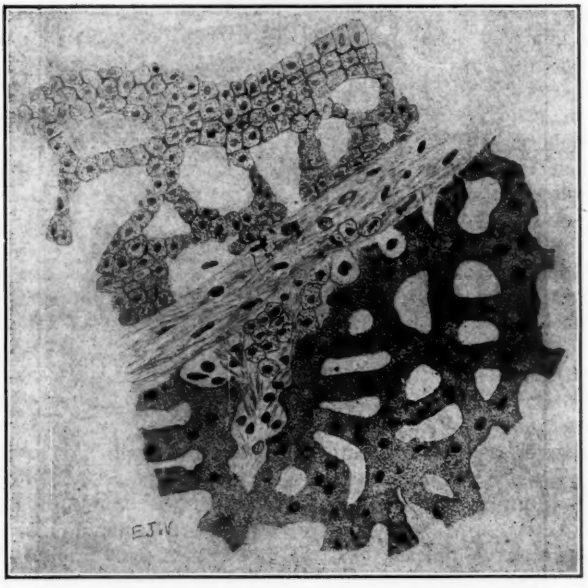
Macroscopic examination of the liver showed no changes except that on the anterior surface there was a small round nodule, white in color, depressed in the centre, and sharply defined.

Between the right lung and the costal pleura there was found some delicate adhesions, which were easily broken up. The base was consolidated and showed a small area of gangrene. No other gross changes were found in any of the remaining organs.

The left kidney was so diseased that it was with some difficulty a suitable specimen could be obtained for the microscopic examina-

tion. The sections were stained with hæmatoxylon and eosin. Only small portions of the tissue here and there would take the stain, the bulk of the section being made up of large homogeneous areas apparently of the type of hyaline degeneration. Occasionally this degenerated material had the appearance of coarse connective tissue, sometimes of the fibrous variety, and again it had a peculiar appearance resembling degenerated muscle fibre. Fig. 1 represents a section taken from the most organized part of the left or diseased kidney. The large T-shaped mass of connective tissue is composed of what appears to be degenerated fibrous tissue, which is homo-

FIG. 2

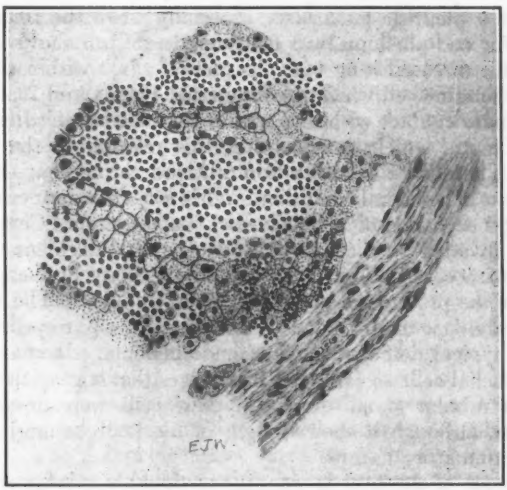


geneous in appearance. Interspersed throughout the fibres are found accumulations of small round cells which take a deep stain with hæmatoxylon. The cells have the appearance of round-cell sarcoma. In smaller numbers is another cell not so well organized which is but little affected by the stain. The greater portion of the section is made up of large cells, usually polygonal in shape, with large nuclei which take a deep stain with hæmatoxylon. The cells are packed closely together and often it is difficult to make out the outline of the individual cell. The protoplasm of the cells is homogeneous and stains but faintly. Between these cells is seen an extensive mass of the same small round cells described above as

occurring in the connective tissue. In addition to this large mass of cells there are several smaller pockets. The large cells are evidently of the endothelial variety. These large cells with occasional small round cells were found here and there in small clusters throughout the hyaline mass of tissue.

Sections of the liver through the small nodule above mentioned were also stained in eosin and hæmatoxylon. The liver cells are found in the majority of instances to be practically normal except near the surface of the organ, where there are few evidences of a fatty degeneration, but this is extremely limited and only found where the endothelial growth is beginning to encroach on the liver tissue. Fig. 2 shows the section of liver in which the capsule is

FIG. 3



thickened and beyond the capsule, but in intimate contact with it is a mass of endothelial cells. This mass of cells makes up the nodule which was described above. These endothelial cells are placed around large irregularly shaped spaces, making a sort of mesh work with no particular pattern. In the sketch the capsule of the liver shows invasion of these endothelial cells. In the liver tissue is a cleft which is filled with connective tissue and with the same endothelial cells. The shapes of the cells vary and apparently the cells adapt their shapes to these open spaces. The cell outline is indistinct. The protoplasm of the cell stains faintly and is homogeneous. The nuclei of the cells are large, well defined, and take a deep stain. The cells closely resemble in many ways the cells found in the kidney; the chief differences are in shape and size.

There is a marked absence of the small round cells which are so abundant in the kidney. There is, however, a cell somewhat larger but otherwise much resembling it which had invaded the liver tissue.

Evidently from the general condition of the organ the growth had made but little headway in the liver. Had the little nodule not have attracted attention there would have been nothing in the general appearance of the organ to suggest a metastasis. The liver was otherwise normal to gross appearances.

The lung tissue showed hemorrhagic infraction. It becomes a question to account for this condition of infarction. Either it was due to an infectious embolus or else to the invasion of the tumor cells. In the light of an absence of any active or healed tuberculous lesion and no previous history of any pleuritic trouble it was natural to suspect a pleuritic metastasis, especially when the frequency of this site for endothelioma was considered. Fig. 3 shows a section through the diseased lung with the pleura. Just within the pleura is noted the same cell which predominated Figs. 1 and 2. This cell is in intimate contact with and within the bloodvessels, and this is the only tissue which demonstrates this relation to the vascular system. There is an extravasation of blood throughout the tissue, being a part of a general condition. What role the tumor cells played in the fatal occlusion of a bloodvessel is a question. Certainly the lung was invaded by the tumor before the fatal accident occurred. It is likewise certain that there was in the body of the patient a source for the production of an infectious embolus. The lung was probably the last organ invaded, and yet in many respects it gave the most typical demonstration of endothelioma. In many places the endothelial cells so arranged themselves that it gave the appearance of a tubular gland. None of these cells were discovered in the pleura, although it showed signs of marked changes but more of an inflammatory nature.

The tumor of the testicle was unquestionably a large round-cell sarcoma, and it is altogether likely that this was the original source of the disease. If this be the case and the metastatic tumors being of the endothelial variety, it is another evidence that endothelioma is a pure sarcomatous tumor.

It is a notable fact that the metastasis was exceedingly slow for a sarcomatous tumor. From the time of invasion to death there elapsed a period of nearly five years.

I am indebted to Dr. C. H. Bunting, of the Johns Hopkins Hospital, for valuable assistance in the microscopic work.

THE EXCRETION OF ACETONE AND DIACETIC ACID IN A CASE OF PERNICIOUS VOMITING OF PREGNANCY.

By Helen Baldwin, M.D.,
NEW YORK CITY.

(From the Laboratory of Dr. C. A. Herter, New York.)

The degeneration in the liver noted by Ewing[1] in cases of pernicious vomiting of pregnancy, as well as in eclampsia, suggested to me that in these conditions there might be found urinary changes due to the breaking down of the proteid molecule and to the associated disturbance of function of the liver cells. The histological alterations in these cases are similar to those in poisoning by phosphorus, where the urine is known to contain pathological organic acids and acetone. It is known also that in phosphorus poisoning the oxidation of the body as expressed by the intake and output of oxygen and by the excretion of urea is greatly diminished. Wakeman[2] has recently shown that in phosphorus poisoning the yield of hexon bases from the proteids of the liver is distinctly diminished, especially in the case of arginin, and it is likely that the damage to the proteid molecule during life (which the changes represent) is related to the diminished oxidations just mentioned.

The similarity of the hepatic lesions seen in phosphorus poisoning and in a late stage of pernicious vomiting of pregnancy thus points to the likelihood that the chemical alterations in the cells are allied and are attended by similar disorders in metabolism. Leucin and tyrosin (products of proteid cleavage) have been found in the urine in both conditions, and are probably present in the livers in cases of pernicious vomiting as well as those of phosphorus poisoning. The following case shows that in the vomiting of pregnancy acetone may be associated in the urine with at least one organic acid, namely, diacetic. Unfortunately, it was not practicable in this case to thoroughly search for leucin, tyrosin, and other amido acids.

This case is interesting not only on account of the finding of these acetone bodies, but also from the evidences which it gives of a high degree of intestinal putrefaction, by the excretion in the urine of large amounts of phenol, indol, and combined sulphuric acid.

The patient was in the care of Dr. Lotta W. Myers, who has kindly allowed me to publish her notes of the case. The history is as follows:

The patient is twenty-six years old. She has never had any

[1] The Pathological Anatomy and Pathogenesis of the Toxæmia of Pregnancy, American Journal of Obstetrics, 1905, vol. li. p. 145.

[2] Zeitschr. f. physiolog. Chemie, 1905, xlix. p. 385.

serious illness. She has always had very severe occipital headaches at the time of her menstrual periods, but has not had attacks of vomiting at that time. She has all her life been very constipated, and states that her stools have always been white; there has not, however, been any peculiarity in the color of her bowel movements during the time that she has been under observation. She was married in November, 1904. The last menstrual period was on January 8, 1905. She began to have nausea about February 5th, but did not have any attack of vomiting until February 20th. At this time she was very constipated. During a long journey in the cars she had a violent attack of vomiting. This was relieved by ordinary treatment, with rest and restricted diet, but on March 4th another very violent attack of nausea and vomiting occurred.

From March 8th to March 30th the patient was kept in bed. The feeding during this time was almost entirely by nutrient enemata. Whenever an attempt was made to begin feeding by mouth a very severe attack of headache, nausea, and vomiting would occur. She had a good deal of pain in the back and of intestinal colic, with flatulence. She had at times great restlessness, with twitching in the limbs, and blurred vision. On March 9th, 10th, and 11th she had four small hemorrhages from the bowels, probably from the rectum.

On March 15th the patient had another severe attack of vomiting. She could not retain any food, and vomited ten times during the day. On March 20th she vomited fourteen times during the day. Her condition became progressively worse. Each attack of vomiting became more alarming than the previous one. On March 30th an operation seemed imperative. She was removed to the Sloan Maternity and an abortion performed by Dr. Cragin. After this the patient made an uninterrupted recovery. The following are the urinary reports before and after the operation:

Urinalysis. March 25, 1905. Volume, 600 c.c.; specific gravity, 1028; reaction acid; urea, 8.86 grams; trace of albumin; no sugar; no leucin; no tyrosin; marked reaction for acetone and diacetic acid; a few epithelial cells and calcium oxalate crystals in the sediment; no casts; the urine was acidified with sulphuric acid and shaken out for organic acids; the organic acid thus obtained from the twenty-four-hour specimen was equivalent to 12.2 c.c. $\frac{N}{10}$ NaOH; indican and phenol both were present in large amounts.

29*th*. Volume, 640 c.c.; clear, light colored; reaction, acid; urea, 8.812 grams; total nitrogen, 4.112 grams nitrogen of ammonia, 0.1954 grams = 4.75 per cent. of total nitrogen; marked reaction for acetone and diacetic acid; marked reaction for indican and phenol; combined sulphuric acid 0.1820; preformed sulphuric acid 0.3531; ratio $\frac{H_2SO_4 \text{ preformed}}{H_2SO_4 \text{ combined}} = 1.9$. Bladder epithelium, a few

leukocytes, and a few calcium oxalate crystals in the sediment; no casts.

30*th*. Twenty-four hours before operation. Volume, 800 c.c.; color, light amber; reaction for diacetic acid more marked than on previous day; large amount of acetone.[1]

31*st*. First day after operation. Volume, 1260 c.c.; distinct reaction for albumin; granular casts; diacetic acid and acetone both present in considerable amount, but the reaction is not as marked as before the operation; large amount of phenol.

April 1*st*. Volume, 1860 c.c.; distinct reaction for acetone; trace of diacetic acid; phenol present, but not as much as before operation.

2*d*. Volume, 1260 c.c.; specific gravity, 1015; no albumin; marked reaction for diacetic acid and acetone; large amount of phenol and indican.

3*d*, 4*th*, and 5*th*. No acetone; no diacetic acid; marked reaction for phenol.

7*th*. Volume, 1080 c.c.; specific gravity, 1025; no albumin; diacetic acid present; trace of acetone; phenol, distinct trace.

8*th*. Volume, 1260 c.c.; specific gravity, 1015; very marked reaction for diacetic acid; trace of acetone; phenol, distinct reaction, but not as much as before. *The patient had a severe headache, with nausea and vomiting on this day.*

9*th*. Volume, 1380 c.c.; specific gravity, 1012; no albumin; distinct reaction for diacetic acid, but not as much as on April 8th; faint trace of acetone; distinct reaction for phenol; preformed sulphuric acid 0.4754; combined sulphuric acid 0.1160.

$$\text{Ratio, } \frac{\text{preformed sulphuric acid}}{\text{combined sulphuric acid}} = 4.0.$$

The patient improved rapidly from this time. No further examination of the urine was made until April 27th, when the report was as follows:

27*th*. Volume, 1000 c.c.; no albumin; specific gravity, 1016; no diacetic acid; faintest trace of acetone; trace of phenol; marked reaction for indican; no casts; bladder epithelium and a few leukocytes in sediment.

28*th*. Volume, 1000 c.c.; specific gravity, 1028; no albumin; no acetone; no diacetic acid; distinct trace of phenol, large amount of indican.

29*th*. Volume, 1000 c.c.; specific gravity, 1018; no albumin; no diacetic acid; faintest trace of acetone; trace of phenol; large

[1] The method used in testing for acetone was as follows: 5 c.c. of strong H_2SO_4 were added to 100 c.c. of urine and distilled. About 60 c.c. of the distillate were collected. A portion of this was warmed in a test tube and rendered alkaline by the addition of a few drops of a solution of potassium hydroxide. Then Lugol's solution of iodine was added drop by drop until there was a slight excess. Then the solution of potassium hydroxide was again added, drop by drop, until the brown color disappeared. With this test, if acetone is present, there is a prompt precipitation of iodoform.

amount of indican; preformed sulphuric acid 0.5444; combined sulphuric acid 0,1008; ratio, $\frac{\text{preformed sulphuric acid}}{\text{combined sulphuric acid}} = 5.4$.

In reviewing the points of interest in this case the most important observation is in the finding of the large amounts of acetone and diacetic acid in the urine. The diacetic acid which was present in large quantities in the specimen of March 25th had increased in the few days before the operation.

While the presence of acetone is not of so much significance as that of diacetic acid, its persistence and increase would indicate that the toxæmia was becoming more grave. The promptness with which the diacetic acid and acetone disappeared from the urine after the operation, and their temporary reappearance after eight days, when the patient had a severe attack of headache and vomiting, are also noteworthy.

The degree of intestinal putrefaction is very striking, as has been noted by others in cases of this kind. Indican and phenol are both present in the urine in large amounts, and the ratio of the combined (aromatic) sulphates to the preformed sulphates, that of 1 to 1.9, indicates an extreme degree of bacterial activity in the intestines.

While the report of a single case is of relatively little value, it has seemed well to publish this record as emphasizing the importance of testing for acetone and diacetic acid in the urine of similar patients.

A PROCEDURE FOR OPENING THE PERICARDIUM.

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It is a well-attested fact that pericarditis is recognized much more frequently at the postmortem examination than during life. That it is difficult to recognize no one will gainsay, as many of the cases develop during the last hours of life as complications of some other diseases; then, some cases which might be recognized are overlooked because the patient is too ill to justify a thorough physical examination. In 4700 cases of the medical service of the Lakeside Hospital there have been diagnosed some 45 cases of pericarditis, of which 13 were fatal—a mortality percentage of 29. In the same service there were 245 cases of pneumonia, with a mortality of 29 per cent., while in 15 cases complicated by pericarditis there was a mortality of 71 per cent., an increase of over 40 per cent. in the complicated cases. There were 158 cases of acute articular rheumatism treated, with a death rate of 3⅓ per cent.; while in 9

cases complicated by pericarditis mortality was 30 per cent. This shows from the clinical side what a marked increase of deaths occur in the two diseases with which pericarditis is most commonly associated, if they are complicated by it.

It may seem that one who has been working along pathological lines is afield in developing an operation, but that is a logical origin. My experience at the autopsy table has impressed on me three points in regard to pericarditis: (1) its great frequency as a complication of other diseases; (2) the comparative ease with which the pericardium may be explored through an opening in the sternum; (3) the necessity of local treatment in severe cases. It seems to me that if advantage is taken of the anatomical relation of the pericardium to the posterior surface of the sternum, there is afforded an easy route of attacking the pericardium and applying local treatment in suitable cases.

The treatment of the pericardium should be the same as that of other serous cavities—viz., joints. In simple conditions, rest and local application of ice-bag or heat. When there is no reaction to this treatment, it is aspirated when there is simple effusion, and opened when pus is present. These procedures should also be applied to the pericardium in similar cases.

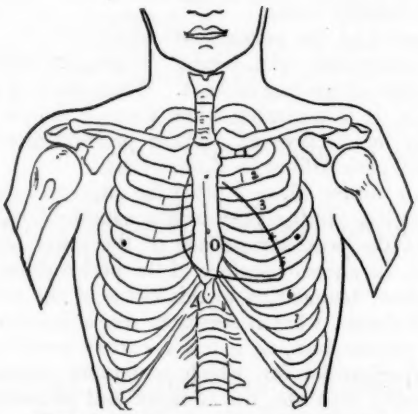
I have been impressed, as I lifted the sternum after severing the ribs on either side during postmortem examination, by the broad connection of the posterior surface of the lower one-third of the sternum with the anterior surface of the pericardium. The pleuræ form the lateral boundaries to this tissue of the anterior mediastinum. This forms, what it seems to me, a feasible route for attacking the pericardium, as it has a correct anatomical basis. A layer of prepericardial fat, which sometimes contains lymphatic glands, normally separates the sternum and pericardium.

In some pathological conditions, as with adherent obliterative pericarditis, fibrous tissue invades this fatty tissue, and then the pericardium is bound to the sternum. The size and shape of this area vary. Voinitch and Sionojensky have worked out a composite picture of the cases in their experience. It forms a triangle, with the base lying to the left of the midline and opposite the fifth intercostal space, and the apex extending down the fifth intercostal space. While I have not worked out a composite picture, I have found but a single case in 110 autopsies that has failed to have a connection that one would miss by going through the sternum at this point. This was in a child aged eight years, with a pigeon-shaped breast. Using this connection as a basis, I have developed the following procedure for entering the pericardium.

A skin incision is made parallel to the mid-line of the sternum and ½ cm. to its left, extending from the level of the upper border of the fifth costal cartilage to the lower border of the sixth. This incision is deepened until the sternum is exposed. This is laid

bare over an area of 2.5 cm. and the periosteum is raised. With a trephine, 2 cm. in diameter, a circular window is made through the bone in the same manner as in trephining the skull. One opens upon the anterior mediastinum and the prepericardial fat then comes to view. The fat is dissected away and the anterior surface of the parietal pericardium is exposed. The pericardium is raised by two mouse-toothed forceps, and an incision is made between them. From this opening into the pericardium, which is at its lowest level, just above its union with the diaphragm, one can readily explore the pericardial cavity, and then institute the treatment that is necessary, according to the conditions present. In children the sternum is narrower, and one may make

FIG. 1



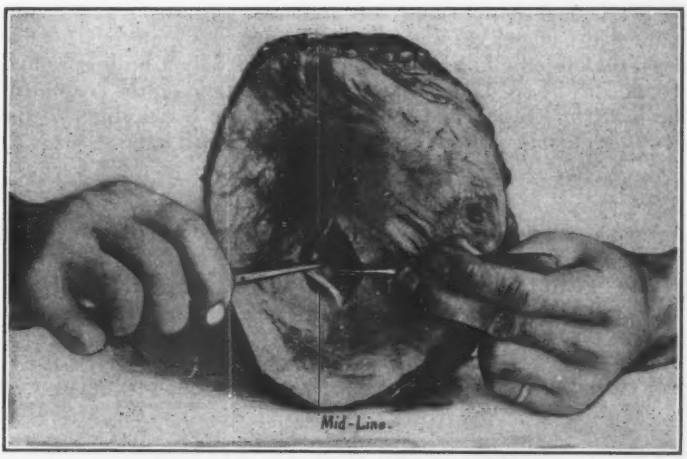
Position of pericardium and window in sternum.

the incision nearer the midline, so that the trephine opening will not separate a rib from the sternum. I have found one case in which the sternum, in the adult, was just 2.5 cm. in width. The internal mammary artery lies from ½ to 2 cm. beyond the sternum, and so it is not injured. The sternum averages about ¾ cm. in thickness in the adult, but varies considerably. I have had one case, and have seen several, in which there was an opening in the sternum at the level of the fifth intercostal space, about ¾ cm. in diameter. This condition is due to a developmental error (lack of fusion of two sides in the embryo).

After one has trephined it is best in those cases where there is pus to fill the cellular portion of the cut edge of the bone with a 15 per cent. iodoform wax, to guard against infecting the bone and to aid in stopping (any excess of) hemorrhage. When the anterior mediastinum is entered one may find some enlarged lymph nodes. I have

found some as large as lima-beans in cases of pneumonia. These should be excised with the fat. When the pericardium is bared it can be identified by feeling the heart beating, and its convexity will be caudal-ward. In those cases where the diaphragm is raised above the normal position by pressure from below, the convexity will be cephal-ward. The pleuræ may be distinguished on either side by the color. In purulent cases it is well to place a purse-string suture before incising the pericardium. A rubber catheter is inserted to aid in flushing and should be left for drainage. Gauze should be packed around the catheter to protect the anterior mediastinum. Better drainage will be afforded if the patient is

FIG. 2



Photograph of specimen. Pericardium held by forceps.

turned on the right side. (The after-treatment will depend on conditions found and their reaction to drainage and flushing with warm saline.)

The advantages presented by this procedure:

1. It is anatomically correct.
2. Easy approach to the pericardium by definite landmarks that are readily made out.
3. The best possible drainage afforded.
4. It permits of ready inspection of the heart and the pericardial cavity.
5. It is devoid of mechanical danger, such as hemorrhage and rupture of the pleura.
6. It can be done under gas anæsthesia.
7. It requires but five to fifteen minutes for its completion.

8. No after-deformity.

9. Least possible danger of spreading infection to the pleural cavities or to the peritoneum.

The advantages and the relative simplicity of the operation are such that the opening of the pericardium should become more frequent. In the height of our present knowledge it seems conservative to treat all cases of purulent pericarditis and large serous effusions, and those cases of fibrinous pericarditis not reacting to rest and the ice-bag by drainage. Cases should be operated upon earlier in the disease before myocarditis has developed from absorption of toxins.

I have performed this operation on eighteen autopsy subjects. Once on a dog, in which I infected the pericardium, manipulated the heart roughly, and simply left drainage to see what would result. Estimated hemorrhage was 5 c.c. The dog was quite sick for five days, then rapidly improved. In two weeks only a healing wound remained. I have made some recent observations on the position of the heart in effusion and the relations of the pericardium to the thorax while under tension, which will be reported later.

THE PERMEABILITY TO BACTERIA OF SURGEONS' RUBBER GLOVES.

By Herbert Fox, M.D.,

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Following a discussion among surgeons as to the absolute impermeability to bacteria of the rubber gloves so generally used in modern surgery, the writers determined to undertake a series of experiments which should determine conclusively whether or not the rubber glove was an absolute safeguard against the implantation of bacteria from the hand of the surgeon to the field of operation.

The fact that pure rubber tissue is impassable to bacteria was not questioned, the doubt hinging rather upon the process of manufacture of the gloves, it being obvious that fissures, minute perforations, or areas of extreme thinness might not infrequently occur, and would of necessity be a prolific source of wound contamination.

Six pairs of gloves of standard make were selected at random from a lot purchased for hospital use, and were subjected to the following experiments:

No. I. The fingers of the gloves were cut off at the web and placed in test-tubes previously filled with peptone broth, high enough for the finger to be immersed about one inch when also filled with bouillon. The free cut edge of the glove finger was tied about the rim of the test-tube, the mouth plugged with raw cotton within the rubber finger, and sterilized in this arrangement for three successive days in the Arnold steam sterilizer. Upon the expiration of the proper sterilization the tubes were separated into two sets of three tubes and one control each. After carefully removing the cotton stopper two tubes were inoculated with the B. coli communis, two with staphylococcus pyogenes albus, and two with streptococcus pyogenes, the control placed under the same conditions, but not inoculated.

After twenty-four hours' growth at 37° C., one tube of each culture was plated by the stroke method from the bouillon without the glove finger. Extreme care was taken in removing the finger that no culture within the rubber should escape. The bouillon in the tubes that had been in contact with the glove finger containing the B. coli communis and staphylococcus pyogenes albus gave a positive culture of the corresponding organism, but not any culture appeared on the tube containing streptococcus pyogenes. There was a possible source of contamination in the two tubes which gave cultures, for, when taken from the incubator, the rubber that had been inverted over the rim of the test-tubes was found loose; it was moist when tied, and dry when it came from the incubator. The same conditions prevailed in those cultures allowed forty-eight hours' growth—*i. e.*, the streptococcus pyogenes negative, staphylococcus and B. coli communis positive. The controls consistently were sterile. If the growth was due to contamination or faulty technique, the reasons for the streptococcus pyogenes' failure to grow were probably incorrect temperature for this particular strain of organism, or unfavorable reaction of the medium. The growth of the second twenty-four hours was at room temperature.

As this was obviously a delicate and difficult method, it was dropped. When the next experiment was done there was no satisfactory strain of streptococcus pyogenes in the laboratory, so the work was continued with the other two organisms.

No. II. This time whole gloves were used, immersed in large flasks of bouillon. The gloves were placed in bouillon about halfway to the wrist, and bouillon poured into three to the same height. The gauntlet was reflected over the rim of the flask and tied tightly. To avoid any risk of external infection a beaker was inverted over the cotton stopper. It may be said here that it is expedient to grease the glove at the surface that comes in contact with the cotton, otherwise the rubber will pull up when the stopper is removed.

After sterilization, as before, the bouillon within the gloves was inoculated with the B. coli communis and staphylococcus pyogenes

albus and allowed to grow forty-eight hours, when cultures were made from within and without the gloves; the former resulted in a pure culture of the germ, the latter being sterile. This experiment was repeated with like results.

No. III. The last test was severe. The flasks and gloves were prepared, stoppered, covered, and sterilized as before, but at the time of inoculation the wrist of each glove was twisted in order to distend the glove, making the pressure greater than when on the operator's hand; then it was tied tightly and the flasks were put at 37° C. for forty-eight hours. The cultures made from the bouillon showed no B. coli communis or staphylococcus pyogenes albus. Entirely new gloves were used each time.

The results of these experiments were such as to justify the writers in arriving at the conclusion that rubber gloves are absolute barriers against wound infection by the hands of the surgeon, in so far that they are absolutely bacteria proof. Any infection occurring must be introduced into the wound from some point not protected by a glove.

THE EFFECT OF EXPERIMENTAL CONDITIONS ON THE VASCULAR LESIONS PRODUCED BY ADRENALIN.

By Leo Loeb, M.D.,

AND

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The pathogenesis of diseases of the bloodvessel walls has been one of the most obscure subjects in pathology, partly because we have been entirely dependent on clinical and anatomical observation for our knowledge of the process; partly because arteriosclerosis and atheroma are found associated with such varied pathological and clinical conditions that it is almost impossible to determine which of these stand in an etiological relation. The experimental production of such changes in the bloodvessel walls is therefore a noteworthy advance in our study of the subject, and the discovery by Crocq and Gilbert and Lion that the injection of bacteria and their toxins may produce such changes was of very great interest. Two years ago the subject was brought into even greater prominence by Josué's discovery that the repeated injection of small amounts of adrenalin into the veins of rabbits produced changes in the aorta which have a close resemblance to atheroma, even calcification being not infrequent. The results obtained by Josué were confirmed by the observations of Rzentkowski, Erb,

Jr., Fischer, Gouget, Pic and Bonamour and others. Experience showed, however, that lesions did not always follow the injections and that the results were influenced by certain unknown factors.

It was with the hope of throwing more light on this obscure subject that the experiments described in the present article were undertaken, the influence of a number of conditions which are supposed to bear a relation to atheroma being studied. Furthermore, this experimental method of producing lesions in the bloodvessels will provide us with means of determining accurately the effect of certain substances believed to be curative in arteriosclerosis of man.

Our experiments may be divided into three groups, as follows, each group being accompanied by a number of control rabbits to which only injections of adrenalin were given.

1. Some experiments were made to determine the effect of excision of the thyroid gland, which was of interest not only because von Eiselsberg has shown that calcification of the aorta may follow this operation in goats and sheep, but also because thyroidectomy seems to have some effect on calcium metabolism and on the healing of fractures, and therefore might have some influence on calcification of the aorta.

2. The influence of lesions of the kidney was worthy of study because nephritis is regarded as one of the most important causes of arteriosclerosis in man, and because interference with the excretion of adrenalin might increase its toxic action. For this reason bichromate of potassium was administered to a series of rabbits in order to produce nephritis; and in a second series one ureter was ligated—this operation not only causing temporary mechanical interference with secretion, but also (as is believed by some investigators) the production of nephrotoxins.[1]

3. As it might be supposed that the injections would have an influence on the structure of developing bloodvessels, and the administration to pregnant animals would also show whether the injections interfered with the course of pregnancy, or whether the latter influenced the toxic action of adrenalin, several pregnant animals were made objects of study.

4. Other questions came up for study during the course of experiments. These were, whether other lesions or results of adrenalin injections had any relation to its toxic action on the aorta, and whether the duration of the experiment, the number of injections, or the amount of adrenalin injected had most influence on the final result.

It has been maintained, especially by Huchard, that increase of blood pressure is one of the most important factors in the production of atheroma, and the thought naturally suggested itself

[1] This view can at present be regarded only as an hypothesis.

that the structural changes following the injection of adrenalin might be connected with its blood-pressure raising property. This led certain investigators to make experiments in this direction. Josué found that nicotin, which causes a rise of pressure, has no effect on the structure of the bloodvessel walls; and L. Braun found that the injection of amyl nitrite with the adrenalin prevents the rise of pressure, but does not interfere with the production of vascular lesions.

Adrenalin chemically seems to be made up of an hydroxyethylamine radical united with a base of orthodioxy benzine. The latter is known as pyrocatechin. Adrenalon, the keton of adrenalin, has been obtained synthetically through the interaction of methylamin and chloracetylpyrocatechin.

As recent studies of Dakin have shown that intravenous injections of 0.002 gram of pyrocatechin cause marked rise in blood pressure, and Baum has shown that its action on the bloodvessels of the skin is similar to that of adrenalin, it seemed worth while to determine whether it has also the power to produce lesions in the aorta. We therefore made a series of experiments in this connection.

1. Influence of Thyroidectomy. The thyroid glands were removed from four rabbits. In three of these the parathyroids were certainly removed also, and as no trace of these could be found in the other (No. 2) at autopsy, we may feel fairly sure that they were removed in this also.

After our experiments had been planned an article by Lortat-Jakob and Sabarianu appeared in which it was stated that after complete thyroidectomy injections of adrenalin produced no changes in the aorta.

Our experiments did not confirm this idea. Only one of the four (No. 3) showed no sclerotic lesions; two of the others showed slight alteration; and in the fourth (No. 2) the entire thoracic aorta was converted into a calcareous tube with a wall like that of the trachea. The three positive results show clearly that removal of the thyroid glands does not prevent the toxic action of adrenalin on the aorta, although it cannot be said that they show that the effect is more pronounced.

2. Influence of Renal Lesions. Potassium chromate was administered by stomach tube to twelve rabbits, in quantities varying from 7 to 40 c.c. of a 1 per cent. solution, over periods ranging from ten to forty days. In all cases the urine was tested at frequent intervals and found to be constantly albuminous. In two of these (Nos. 2 and 22) the chromate was administered during the latter part of the experiment only. In four other rabbits the left ureter was tied close to the kidney, and at autopsy occlusion was found to be complete in each case. In the latter injections of adrenalin were given twice a week until death, which occurred from twenty-

two to forty-seven days after operation. The only one (No. 5) which showed any aortic lesion lived twenty-seven days.

On the whole, the lesions were no more severe in these rabbits than in corresponding controls, which seems to show that interference with the elimination of adrenalin does not increase its toxic action. This agrees with the results obtained by Embden and v. Fürth; according to the latter investigators the blood-pressure raising power of adrenalin is quickly destroyed in the blood, and it also agrees with the fact shown by Cybulski that its effect on blood pressure was as transitory after double nephrectomy as before.

3. Influence of Pregnancy. Six rabbits were treated during the whole, or almost the whole, of pregnancy—some almost every day. In three of these no alteration of any sort was found in the aorta; in one of them there was a small spot of certain change; in one slight and doubtful lesions were found, and in the last these were fairly marked, but as injections were continued about four weeks after delivery this case must be excluded when we are considering the influence of pregnancy on the lesions produced by injections of adrenalin.

It was evident that the course of pregnancy was not influenced, and the young, which were seen in three cases, appeared to be quite healthy. Several of the offspring from two births were dissected, but no lesion of the arteries or other abnormality was to be found.

We may therefore state: 1. Adrenalin injections do not interfere with the course of pregnancy or delivery in rabbits. 2. They have no effect on the development of the vascular system of the fetus. 3. It seems probable that pregnant animals are less susceptible to the toxic action than are others. The latter conclusion cannot yet, however, be regarded as a definite one.

4. Relation of Certain Other Lesions. In some rabbits aortic changes occur after a very short time and from very small amounts of adrenalin. Thus, Rzentkowski reports lesions after four days, and only a little more than 0.5 c.c. of the 1:1000 solution, and Josué after eight days. Several of our animals also showed very early results. No. 10 after five injections of 0.15 c.c. each, No. 32 after six injections of 0.2 c.c. each, and Nos. 24 and 5 after seven and eight injections, respectively, of 0.15 c.c. each.

All these cases and one other (No. 4, in which very pronounced calcification was found) showed inflammatory changes in the lungs and pleura, and some in the pericardium also. Only one rabbit, No. 28, showed similar pulmonary lesions without atheroma, and it differed from the others in the fact that it did not lose weight during the experiment, and seemed in good condition until the end.

As injections of adrenalin are known to cause œdema and punctate hemorrhages in the lungs, we may, with some degree of probability, connect the pulmonary lesions with the injections, and

may draw the following conclusions: Either (*a*) pneumonia and pleurisy favor the development of sclerotic changes in the aorta, or (*b*) those rabbits which are most susceptible to the toxic action on the lungs are most liable to its effects on the aorta.[1]

It also appeared that those rabbits which lost weight showed vascular lesions sooner and to a more marked degree than those which remained in good physical condition. This may be either because systemic disturbance makes the animals more susceptible to the action of adrenalin, or because the latter interferes with the general metabolism of those animals in which its local action is most manifest. Of the fifteen animals which lost weight, ten showed aortic lesions; while of the seventeen which did not, only seven showed such—41 per cent. as compared with 66 per cent.

5. INFLUENCE OF PYROCATECHIN. This substance, dissolved in normal salt solution, was injected intravenously in steadily increasing doses in a series of four rabbits. Only one of them died during the course of the experiment, and as it received less than one-tenth as much as the others, it seems probable that the death was due to some other cause.

The injections were begun with only 0.2 c.c. of a 1:1000 solution, but were slowly increased to 2 c.c. of a 1:100 solution. Doses of 1 c.c. of this solution and upward caused clonic convulsions, and three rabbits received twenty-two such injections each. In spite of the enormous dosage the three rabbits retained their health and weight to the end.

In the rabbit which died early in the experiment (after thirty-one days and sixteen small injections) and in one of the others there were pinhead-size spots which may have been beginning aortic sclerosis, but were probably not. The others showed no sign of any lesion.

We may thus conclude that if pyrocatechin has any deleterious action on the walls of the bloodvessels it is extremely insignificant and not at all to be compared with that of adrenalin.

These results agree with those mentioned in regard to the etiological significance of high blood pressure, and suggest that experiments with a series of bodies closely related to adrenalin, but not causing any increase in blood pressure, might materially advance our knowledge of the mode of its toxic action.

Forty rabbits were used in these experiments, but four of these died too early to be of value, and four were used for pyrocatechin. Of the remaining thirty-two, seventeen showed aortic lesions and ten had calcification; fifteen showed no result. The average duration of the thirty-two experiments was thirty-one days; fourteen lived longer than thirty days, and of these, eleven showed sclerosis;

[1] It is intended to determine later whether lesions of the lung occurring in rabbits not injected with adrenalin are likewise connected with aortic changes.

eighteen died within thirty days, and of these only six showed changes; sixteen received more than 3 c.c., and of these, eleven showed lesions; sixteen received less, and of these, six showed lesions. In the former case 78.6 and 33⅓ per cent.; in the latter 69 and 37.5 per cent. This shows the greater effect of time as compared to amount of adrenalin.

It is perhaps not fair to make direct comparisons between the different series on account of the fact that the ureter ligation rabbits all died within twenty-eight days, and all the thyroidectomy rabbits and pregnant rabbits lived beyond that time. The chromate rabbits lived on the average nineteen days, and the controls thirty-five days. This largely explains the reason that a larger percentage of the thyroidectomy rabbits and controls showed lesions than of the chromate and ureter ligation series.

In generalizing on the whole series of experiments we would like to draw attention first to the fact that although the relation of a number of the factors has been determined there still remains a marked variability in the reaction which we cannot yet explain. The possibility must, of course, be considered that different samples of adrenalin solution varied in toxic power, but even animals injected at the same time with the same solution showed extreme differences. This individual variation may also show itself in the reaction to a single injection. Thus, in one rabbit fatal œdema was caused by the injection of 0.3 c.c., while in other cases as much as 1 c.c. followed by only slight pulmonary œdema.

In one case, after a rabbit had ceased breathing and the heart action was no longer perceptible, rhythmic pressure on the chest wall caused a complete and surprisingly rapid recovery. Whether this was due to artificial respiration or to massage of the heart it is impossible to say.

As positive factors in determining the amount of reaction we can state with some certainty that:

1. Injections given at intervals of four days have more effect than the same number of injections of the same size given closer together.

2. Granted the injection of a certain minimum dose which is able to cause aortic lesions, even a marked increase in the size of each dose causes little, if any, increase in the toxic action. The increase, if any, is markedly out of proportion to the amount injected.

3. Injections given every four days have almost as much effect as injections given every day for the same length of time.

Thus, we see that in determining the result the length of time is the most important factor, the number of injections much less, and the size of the dose still less important. We give these latter conclusions with a certain reserve, as the factor of individual susceptibility complicates the effect of the injections.

Our results agree with those of other observers that the lesions

are limited almost, if not entirely, to the aorta. In most cases patches were found in the arch and descending thoracic. In four cases (Nos. 5, 13, 30, and 32) the abdominal was also involved, and in one of these (No. 13) the change in the thoracic was minimal or doubtful. In No. 27 there was only one small placque, that being in the descending thoracic. In four cases (Nos. 1, 4, 22, and 29) the only lesion was in the ascending portion or arch. In one of these (No. 1) the only spot was in the sinus of the valsalva. In No. 22 there were two small aneurysms.

Microscopic sections were made of aortas in different stages of degeneration, fixed in formalin and stained by hæmatoxylin and eosin, and by Weigart's elastic-tissue stain.

In early cases we find only loss of nuclei in the muscle cells of the media over larger or smaller areas. These cells may be partly vacuolar. In one specimen there were signs of amitotic division of nuclei at one place near the adventitia. In a few sections the internal elastic membrane seemed to be split into a number of fine lamellæ. Whether this has any connection with the other changes and is of any significance only further study can show, as in advanced cases it was still well preserved.

In more advanced cases impregnation with lime salts was found in the central part of the media in the form of larger or smaller plates. The nuclei of the muscle cells had entirely disappeared. The elastic fibres may be well preserved, but they generally loose their wavy appearance and run straight through the calcified area. They may be pushed together as if the substance separating had been lost, and may even look as if glued together. In other cases they begin to disintegrate and may almost disappear. These changes in the elastic fibres may occur in areas near calcareous plates where calcification itself has not yet begun. At some points ruptures had occurred in the incrusted tissue and the elastic fibres with the tissue between was bent into the shape of a shepherd's crook. Changes in the intima and in the adventitia may be absent or very slight even in advanced cases.

Inflammatory processes were never found. There was no round-cell infiltration in any of the coats and no new formation of blood-vessels or connective tissue. This absence of changes of an inflammatory nature is characteristic of the early stages, but collections of certain cells may be found sometimes in later stages around the calcareous plates, as described by Erb. We cannot exclude that new formation of connective tissue may, perhaps, occur in the intima, but it certainly is not constant, as it was not found in our specimens.

We see, therefore, that the character of these lesions differs essentially from the findings in arteriosclerosis and atheroma in man, where the so-called inflammatory changes are very prominent, and where, according to some observers, the initial change is in

the elastic membrane and degenerative processes in the intima are of more importance than those in the media.

Although the difference in histological appearance between atheroma in man and the changes in the aorta of rabbits treated with adrenalin is so great, we cannot positively state how fundamental the differences are or whether they may not after all be of only secondary importance.

ABSTRACT FROM PROTOCOLS.

Series I. Thyroidectomy.

1. Weight, 2260 gr.; at death, 2080 gr. Operation four days before first injection. Duration forty-three days. Adrenalin nineteen injections; 3.9 c.c. Killed. Autopsy showed small diseased areas in the thoracic aorta and one calcareous patch in the sinus of Valsalva.

2. Weight, 2100 gr.; at death, 1760 gr. Operation nineteen days before first injection. Duration fifty-three days. Adrenalin twenty-eight injections; 6.4 c.c. Killed. Autopsy showed extreme calcareous change in the entire descending thoracic aorta. This case shows especially well the fact that thyroidectomy does not prevent the action of the adrenalin, it being, perhaps, our most marked case.

3. Weight, 1960 gr.; at death, 1900 gr. Operation ten days before first injection. Duration fifty-three days. Adrenalin twenty-eight injections; 6.4 c.c. Killed. Autopsy showed no alteration in the aorta. This is especially interesting, as it agreed in all details with No. 2, but showed such a very marked difference in the final result. It shows especially well the danger of trusting to conclusions drawn from a small number of investigations.

4. Weight, 1620 gr.; at death, 1800 gr. Operation seven days before first injection. Chromate begun on twenty-first day. Six injections; 37 c.c. Duration fifty-three days. Adrenalin twenty-eight injections; 6.4 c.c. Died. Autopsy showed pneumonia of lower right lung. Slight disease of ascending thoracic. This case is especially interesting as it bears on several points. In the first place, although it died of pneumonia, it did not show marked lesions; but this fact seems to be counteracted by the remarkable gain in weight. Finally, it shows the lack of effect of the chromate.

Series II. (A) Ureter Ligation.

5. Weight, 1700 gr. Operation four days before first injection. Duration twenty-five days. Adrenalin eight injections; 1.2 c.c. Died. Autopsy showed right-sided pneumonia, bilateral pleurisy, and fibrinopurulent pericarditis. Also an apparently degenerative (fibrous?) patch in the wall of the heart. Aorta diseased throughout. Extreme calcification of the descending thoracic, which affected

somewhat the origins of the intercostals. This case is of extreme interest. It is the only one of this series which showed any lesion in the aorta, but as this was so very extreme and occurred after so small an amount of adrenalin, it emphasizes again the difference in individual reaction. The association of pulmonary lesions is of great interest. The fact that no other of this series showed vascular changes makes it probable that the ligation of the ureter had nothing to do with the extreme alteration. The weight at death was, unfortunately, not taken.

6. Weight, 1400 gr. Operation three days before first injection. Duration forty-three days. Adrenalin twelve injections; 1.8 c.c. Killed. Autopsy negative.

7. Weight, 1600 gr. Operation four days before first injection. Duration nineteen days. Adrenalin five injections; 0.75 c.c. Died. Autopsy negative.

8. Weight, 1800 gr.; at death, 1420 gr. Operation three days before first injection. Duration twenty-five days. Adrenalin seven injections; 1.05 c.c. Died. Autopsy showed doubtful or minimal change in the aorta. In spite of the great loss of weight this rabbit did not show itself susceptible. (See remarks on No. 24.)

Series II. (B) Chromate of Potassium.

9. Weight, 1900 gr. Chromate 1 per cent. Nine injections; 20 c.c. Duration thirteen days. Adrenalin four injections; 0.6 c.c. Died. Autopsy showed no lesions.

10. Weight, 2200 gr. Chromate 1 per cent. Ten injections; 30 c.c. Duration sixteen days. Adrenalin five injections; 0.75 c.c. Died. Autopsy showed double pneumonia and pleurisy. Aorta diseased with calcified plates in the thoracic portion. This is the smallest amount of adrenalin which gave rise to lesions in the course of our experiments and pulmonary lesions were found.

11. Weight, 1400 gr.; at death, 1200 gr. Chromate five injections; 25 c.c. Duration twenty-seven days. Adrenalin ten injections; 1.5 c.c. Killed. Autopsy showed one small calcified spot in the arch and a large diseased patch along the descending thoracic aorta. (See remarks on No. 23.)

12. Weight, 1340 gr. Chromate four injections; 15 c.c. Duration fourteen days. Adrenalin five injections; 0.8 c.c. Died. Autopsy showed no vascular lesions.

13. Weight, 1830 gr.; at death, 1660 gr. Chromate eight injections; 40 c.c. Duration thirty-eight days. Adrenalin fourteen injections; 3.2 c.c. Killed. Aorta showed minimal changes in the thoracic aorta and considerable calcification in the abdominal. This is controlled by No. 17, which showed no pathological changes and which has been pregnant.

14. Weight, 1900 gr. Chromate five injections; 7 c.c. Duration

nine days. Adrenalin four injections; 1.1 c.c. Died. Autopsy showed no lesions of aorta.

15. Weight, 1400 gr. Chromate three injections; 12.5 c.c. Duration ten days. Adrenalin five injections; 1 c.c. Died. Autopsy showed no vascular lesions.

16. Weight, 1740 gr.; at death, 1800 gr. Chromate six injections; 65 c.c. Duration twenty-nine days. Adrenalin twenty injections; 13 c.c. Killed. Autopsy showed no vascular lesions. Interesting on account of the large amounts of adrenalin and chromate and the absence of lesions.

Series III. Pregnancy.

17. Weight, 2060 gr.; at death, 2240 gr. One day pregnant before first injection (?). Duration thirty-two days. Adrenalin eighteen injections; 3.85 c.c. Killed. Showed signs of recent delivery. Autopsy showed no vascular lesions. Injections were started at about the beginning of pregnancy and were given at first every other day, later, every day. She was killed because of a sudden loss of weight, which had been above 2300. The absence of lesions in spite of large amounts of adrenalin is noteworthy.

18. Weight, 2780 gr.; at death, 2720 gr. Two days pregnant before first injection (?). Duration thirty days. Adrenalin seventeen injections; 3.7 c.c. Killed on account of loss of weight, which had risen to 3060. Autopsy showed one small placque in aorta. Remarks as for No. 17.

19. Weight, 2700 gr.; at death, 2550 gr. Five days pregnant before first injection (?). Duration twenty-two days. Adrenalin nineteen injections; 7.8 c.c. Killed on account of loss of weight, but found to be still pregnant and about five days from term. Autopsy showed no calcification; descending aorta appeared uneven. Fetuses normal.

20. Weight, 2000 gr.; at death, 1900 gr. Four days pregnant before first injection (?). Chromate begun early. Seven injections; 20 c.c. Duration twenty-six days. Adrenalin thirteen injections; 3 c.c. Killed after birth of three normal young. Autopsy showed no lesion in mother or young.

21. Weight, 1800 gr.; at death, 1660 gr. Four days pregnant before first injection. Chromate begun early. Six injections; 65 c.c. Duration twenty-nine days. Adrenalin twenty injections; 13 c.c. Killed on account of sudden loss of weight, which had been 1850. Uterus recently pregnant. Autopsy showed no vascular lesions.

The above cases are interesting as showing the protective influence of pregnancy and the negative effect of chromate.

22. Weight, 2000 gr. Eight days pregnant before first injection. Chromate begun only a week before death. Two injections; 10 c.c. Duration sixty-six days. Adrenalin twenty-one injections; 3.9 c.c.

Young born twenty-two days after beginning injections. They appeared normal, but were eaten by the mother before they could be autopsied. Died. Autopsy showed one small calcareous patch in the descending arch of the aorta associated with a small aneurysm. It may be noted that the chromate was not of sufficient duration to have any influence. The presence of calcification in spite of pregnancy is, of course, discounted by the fact that injections were continued six weeks after delivery. This was our longest continued case, and, in spite of that fact, showed only slight lesions.

Series IV. Controls.

(*a*) Controls in good health.

23. Weight, 1620 gr. Killed as control to No. 11. Duration twenty-seven days. Adrenalin ten injections; 2.1 c.c. Autopsy showed no vascular lesions. Although this received rather more adrenalin than the corresponding chromate rabbit, it showed no lesions, while that showed vascular changes.

24. Weight, 1700 gr.; at death, 1350 gr. Killed when very sick as control to No. 8. Duration twenty-four days. Adrenalin seven injections; 1.5 c.c. Autopsy showed double pneumonia. Aorta showed calcification of the entire thoracic aorta, most marked in the descending part. Showed much more marked lesions than the corresponding rabbit with ligated ureter.

25. Weight, 1600 gr.; at death, 1460 gr. Killed as control to No. 22. Duration sixty-five days. Adrenalin twenty-one injections; 3.9 c.c. Autopsy showed calcification of the arch and of the abdominal aorta, and disease of the entire vessel. Comparison shows perhaps the protecting influence of pregnancy in No. 22, which had much less marked lesions.

26. Weight, 1660 gr.; at death, 1500 gr. Killed as control to thyroidectomy rabbits. Duration fifty-one days. Adrenalin twenty-two injections; 5.1 c.c. Autopsy showed pathological change in the arch of the aorta, but no calcification. It agreed fairly well with the average of the thyroidectomy series.

27. Weight, 1700 gr.; at death, 1960 gr. Killed as control to Nos. 16 and 21. Duration thirty-two days. Adrenalin nineteen injections; 11.2 c.c. Autopsy showed beginning disease of the descending thoracic aorta. The corresponding rabbits showed no lesions.

28. Weight, 1460 gr.; at death, 1700 gr. Duration eighteen days. Adrenalin nine injections; 2.7 c.c. Died. Autopsy showed pneumonia and pericarditis. Aorta showed no lesions. This case should be compared with No. 4. Both increased in weight and showed little effect from the adrenalin, although they died of pneumonia.

(*b*) Controls in poor condition originally.

29. Weight, 1410 gr.; at death, 890 gr. Control to No. 13. Duration thirty-five days. Adrenalin fourteen injections; 3.1 c.c. Died.

Autopsy showed five or six small patches in the upper part of the aorta.

30. Weight, 1300 gr. Lost weight, but no exact record. Duration thirty-four days. Adrenalin ten injections; 1.5 c.c. Died. Autopsy showed abscesses under the skin of the abdomen. The wall of the left ventricle appeared degenerating. Aorta, about twelve calcareous patches in the thoracic and one in the abdominal. Almost similar to No. 5, which showed about equally severe lesions. Interesting as compared to No. 29, as it was so much more affected by less than half the amount of adrenalin. A case in favor of the view that duration is of more influence than anything else.

31. Weight, 1240 gr.; at death, 1140 gr. Duration twenty-one days. Adrenalin twelve injections; 3.8 c.c. Died. Autopsy showed no vascular lesions.

32. Weight, 1520 gr. Lost weight, but no exact record. Duration ten days. Adrenalin six injections; 1.3 c.c. Died. Autopsy showed disease of the arch and nodule of consolidation in apex of lung, and fibrinous pericarditis. The latter changes explain probably the aortic lesions found ten days after the beginning of the experiment.

Series V. Pyrocatechin.

33. Weight, 1300 gr. Killed. Weighed at death 1500 gr. Duration fifty-seven days. Pyrocatechin thirty-eight injections; 288 c.c. Killed. Autopsy showed no aortic lesions. Gained in weight, notwithstanding the large doses of pyrocatechin.

34. Weight, 1920 gr. Killed. At death, 1740 gr. Duration fifty-seven days. Pyrocatechin thirty-eight injections; 288 c.c. Killed. Autopsy showed no aortic lesions.

35. Weight, gr. 1560 gr.; at death, 1310 gr. Duration thirty-one days. Pyrocatechin sixteen injections; 19.3 c.c. Killed. Aorta showed a few pinpoint-sized opaque spots which were possibly not of the same character as the vascular lesions caused by adrenalin. Animal had lost much weight. Lungs showed scattered areas of solidification.

36. Weight, 1650 gr.; at death, 1625 gr. Duration fifty days. Pyrocatechin thirty-three injections; 258 c.c. Killed. Aorta showed one pinpoint-sized opaque spot which was possibly of the same character as the ones caused by adrenalin.

References.

The work of some of the authors mentioned below is of such recent date that only references of reports on communications made to societies could be given.

Crocq. Archives de méd. exper., 1894, tome vi. p. 583.
Gilbert and Lion. Ibid. 1904, tome lx. p. 73.
Josué. Presse médicale, 1903, tome x. p. 796.
Josué. Compt.-rend. Soc de biol., 1904, tome lvii. No. 35.

Rzentkowski. Berlin. klin. Woch., 1904, Bd. xli. p. 830.
Erb, Jr. München med. Woch., April 25, 1905, p. 829.
Fischer. Ibid., Bd iii., No 1, p. 45; and Deutsche med. Woch., 1905, No. 17, p. 693.
Gouget. Compt.-rend. Soc. de biol., December 19, 1903.
Pic and Bonamour. Ibid., February 10, 1905, No. 5.
v. Eiselberg. Deutsche Chirurgie.
Josué. Bulletin médicale, 1904, No. 88, p. 956.
Dakin. Journal of Physiology, 1905, vol. xxxii.
Baum. Berlin. klin. Woch., 1905, Bd. xlii.
L. Braun. München. med. Woch., 1905, No. 11, p. 533.
E. Friedmann. Hofmeister Beiträge, 1904, vol. vi.
Lortat-Jakob and Sabarianu. Compt.-rend. Soc. de biol., 1904, tome lvii. p. 444.
Embden and v. Fürth. Hofmeister Beiträge, 1903, Bd. iv.

A STUDY ON BRACHIAL BIRTH PALSY.[1]

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HISTORICAL INTRODUCTION AND CRITICAL DIGEST OF THE LITERATURE OF BRACHIAL BIRTH PALSIES (L. P. CLARK).

Notwithstanding the fact that Duchenne first described brachial birth palsy due to traction in his work on localized electrization in 1872, there is evidence that arm palsies occurred in the process of delivery nearly two hundred years ago. Indeed, a careful review of obstetric works of a still earlier date would probably disclose cases of brachial birth palsy. The palsy is undoubtedly as old as forcible intervention in obstetric practice. Indeed, the association of the two facts early presaged the cause and effect relationship which is known to exist between them in our own time.

A case of compression paralysis of the arm is recorded in an old obstetric report as early as 1768 (Smellie). The labor in which it occurred was a face presentation. Owing to prolonged compression to which the child had been subjected both arms remained paralyzed

[1] Grateful acknowledgment is made to Dr. E. A. Sharp for photographs of the cases, and to M. Peterson for the anatomical drawings of the operative field. A preliminary report was made before the Neurological Society of New York in April, 1904. A special paper on the pathology was presented before the same society in November, 1904. This paper reports the cases only to January, 1905. Final results will be reported in a future communication.

for several days. Discussions and theses at an early date bear testimony of the needs of classification of the causes and the resultant types of obstetric birth palsies. Thus some cases which apparently showed a wide range and severe degree of palsy at birth recovered in a few days, where others of slighter apparent defect recovered but little or not at all after years of medical treatment. A significant case of the former type is that of Jacquemier (1846), in which the palsy of the upper arm recovered in twenty days, while the first case of Duchenne-Erb palsy reported by Danyon in 1851 illustrates the severe destructive nature of the typical lesion. In this case, after an apparent simple compression which induced the palsy, the child died on the eighth day. A profound and extensive hemorrhagic infiltration was found in the entire brachial plexus. The condition found at autopsy was strongly suggestive of a lacerating lesion of the brachial plexus. Still other cases antedating Duchenne's report are those of Doherty (1844), Mattei (1862), Guenoit (1864), and Depaul (1864). The case by the two latter authors was reported in 1864 by each separately, and, owing to error of subsequent writers, has done double duty, being counted as two cases. Although compression was alleged as the cause, the unfavorable outcome leads one to suspect that it too, as in Danyon's case, was the result of a stretching and lacerating of the plexus. While Duchenne recognized three forms of brachial palsy in the newly born, he expressly outlines the clinical syndrome of the type classed in this study as "laceration brachial birth palsy."

(In this study the cases are classified as (1) "compression birth palsy," the result simply of pressure; and (2) "laceration birth palsy," from overstretching of the nerves with resulting laceration in varying degrees of the nerve sheaths and their contained vessels as well as of the nerve fibres themselves.)

Duchenne saw a few cases of compression palsy and relatively few of a subspinous dislocation of the humerus. As Duchenne had no definite conception of the nature of the nerve lesion present in any of these palsies, a more exact determination of the relationship of the different causes and effects was not possible at that time. He had no doubt, however, that in his five cases of obstetric palsy traction was the sole factor in its production, inasmuch as no forceps were used in the delivery and great pulling of the head by hand was employed in the process of extraction. Two of his cases were breech presentations. Duchenne saw all of the five cases in consultation with the obstetricians in whose practices these nerve accidents occurred.

Immediately following Duchenne's work upon these conditions, four cases were reported by Nadaud, including a case each of Bailly's, Tarnier's, Chantreuil's, with one of his own.

After Erb and others had described this same type of paralysis in adults, cases of obstetric palsy were reported by Schultz, Schu-

macher, Kustner, Thorburn, Ballenhagen, Hochstetter, Fieux, and many others. (See appended bibliography.)

Following Duchenne's work, Seeligmüller (1874) presented the next case of laceration brachial birth palsy. Although Erb (1874) reported one case of this type in his original presentation of the adult lesion, the case was not published until after that of Seeligmüller. A fact of greater interest than the simple report of cases at this time was brought up by Fritsch, who in the discussion of Erb's thesis cites a case of arm paralysis in a child two days old, associated with a hæmatoma at the lower portion of the sternomastoid muscle (Erb's point). The case possibly foreshadowed the hemorrhagic nature of the lesion, which until in the present thesis has never been mentioned in the literature of the subject. If so, it must have been a very gross form of the lesion, as the cases here presented gave no history of such palpable birth injury. Moreover, the paralysis in Fritsch's case disappeared with the absorption of the hemorrhagic mass. The accident occurred in a head presentation with forcible forceps delivery. The case is all the more interesting as it was but a single illustration of a series studied by Fritsch in connection with hemorrhagic extravasations in this locality. He had repeatedly seen superficial hæmatoma about the shoulder in breech cases and had inferred that deeper lesions of the same hemorrhagic character should have existed. With the exception of the case mentioned above, his series consisted of stillborn infants; therefore, paralysis could not appear. No histopathological study of the lesion was made. Had this been done the exact nature of the lesion in laceration brachial birth palsy must have long since been demonstrated. The gross appearance of hemorrhage mentioned by Fritsch was not present in either Erb's or Seeligmüller's cases, although the latter was a bilateral lesion of the plexus, occurring after a difficult extraction. No traumatism or complications were associated in the case. Seeligmüller (1877) reported three cases of lower arm type of brachial birth palsy, in which a paralysis complementary to that of Erb was present—*i. e.*, paralysis of the muscles supplied by the lower roots of the plexus. The palsy was associated with ocular symptoms such as narrowing of the pupil and palpebral fissure on the same side as the lesion. This type of brachial palsy was first described in the adult by Flaubert (1827). In Seeligmüller's case the infant was born unaided, but after a very protracted labor. Probably the lesion was purely a pressure one, as the child completely recovered in a few weeks. It is of interest to note that Erb's single case of laceration brachial birth palsy reported in 1874 and published in 1877 was thought to be due to pressure of the hooked finger in the axilla, as practised in the Prague method of version and forcible extraction. The inconsistency of a permanent palsy (over two years old at time of report) being induced in the fifth and sixth nerve root by pressure in the axilla was apparently not appre-

ciated by Erb, notwithstanding the fact that his thesis was specially directed toward proving that his type of adult palsy was caused by none other than pressure of the clavicle upon the fifth and sixth cervical roots in the neck.

Nothing essentially new is contained in the report of cases of Bernhardt (1880) and Thorburn (1886). The first monographic consideration given to the subject was that of Hamond (1881); but the work sheds no new light upon the nature of the lesion. The same may be said of Roullaud's (1887), although a number of new cases were reported. The elaborate work of Kustner (1889) still repeated the dictum that the persistent Duchenne's obstetric palsy must be secondary to bone injuries, especially fracture at the upper epiphysis of the humerus. The absence of all subsequent evidence of a birth fracture in these many cases did not seem to have rendered the author any the less certain of the etiology of the palsy. He believed, however, that the cases which recovered spontaneously were due to forceps compression. No mention is made of stretching or rupture of the plexus. This reactionary view appeared to suppress research work for some time, as but little was done on the subject in the next few years. Schultz published the first article directed specifically to pathogenesis. Erb's theory was generally accepted at the time. Schultz reported a case of arm paralysis in a child seen when two years old, with a history of breech extraction and bringing down of the right arm behind the head. The case was typical of the laceration palsy herein described. Schultz applied Erb's theory of clavicle compression and elaborated a defence of this view. The inaugural dissertation of Arens (1889) at Göttingen upon "Obstetric Paralysis" is worthy of passing mention, as it was a monographic treatment of the subject at the hands of an American. But one new case was reported. It was a breech presentation delivered by the Veit-Smellie method. A bilateral Duchenne-Erb palsy resulted. The left side recovered rapidly. Arens assumed the presence of a deep-lying hæmatoma on the left side which, as recovery took place, underwent absorption. He conceived that the right plexus had sustained a more severe injury (rupture?). There appears to be a gap in the literature between 1889 and 1897, the date of Fieux's essay upon the etiopathology of obstetric palsies. The experimental work of this author leads him to depart strongly from Erb's theory of compression. He believes that overstretching is the chief causal factor. (The experimental work on the etiology described later was done and the conclusions formulated before the thesis of Fieux had been discovered in the literature.)

During this quiescent period (1889-1897) Comby and Jaffroy (1891) reported cases, as did also several American authors (Lovett and Burr, 1892). Burr's report ignored all the work previously done, as well as that of Lovett, which was published in the same volume of *Transactions of the Boston Obstetrical Society*. Burr

styled the disease under a new name, "spinal paralysis," and regarded it as due to a "rare type of anterior poliomyelitis." The discussion failed to correct the diagnostic error. Ten cases were reported under this head, which were all typical of laceration brachial birth palsy. A paper of extreme interest is that of Guillemot (1897), in which he reports a veritable epidemic of obstetric birth palsies in a particular locality covering a term of years. There were no less than 30 cases in all delivered by the same midwife by podalic version. Guillemot saw 12 of these cases, most of whom suffered from the permanent or laceration type of palsy. They were all adolescent or adult cases. Two of the cases were bilateral in type. In his research 2 cases in each of two families were noted. In those days no particular explanation seems to have been expected from the obstetrician to account for any of these accidents. A sufficient number of cases had been reported by 1897 for Cibert to collect a series of 66, but nothing new was brought out. Studies upon the different types of obstetric palsy soon began to appear. Seeligmüller and Thorburn reported the "Klumpke paralysis" (1885), which should properly be called after Flaubert, who first described the condition in 1827. The type consists of a lower brachial plexus lesion. In 1896 Weil published a valuable article upon the differentiation of superior arm, lower arm, and mixed-arm types of birth palsies and their various combinations in the same case. He gives a very clear account for the first time of an entire cervicobrachial lesion.

In relation to the possible extent of the lesion, aside from that in the plexus nerves, the work of Barbarosa di Grovellona may be mentioned here. The possibility of there being a cord lesion associated as the result of the heavy traction employed in delivery is worthy of attention. Barbarosa places on record an entirely new form of obstetric palsy. The brachial paralysis he reports is accompanied by cord lesions. The cases illustrating this type consisted of double brachial monoplegia and amyotrophy as a result of the cord lesion (?). The condition was not common. It would seem that an error of diagnosis had been made. He inferred that the violent stretching in laceration birth palsy may exceptionally produce a tearing of nerve roots at or in the cord itself. The possibility of this occurrence will be dealt with in the experimental work on etiology in this research.

Inaccurate as the early statistics must have been it is of interest to note that of the 95 cases collected by Schümacher (1899), 53 were head and 40 breech cases; of the 53 head cases, forceps were used in 28; spontaneous births, 10; history of finger hooked in axilla, 10; history of traction with head laterally inclined, 8. Of the 40 breech cases 6 were of spontaneous delivery and 34 assisted.

In a careful review of the literature it is evident that with very few exceptions obstetric palsy is due to manipulation in delivery.

Obstetric palsies form a class entirely by themselves and have nothing to do with cerebral or other congenital palsies. As a rule, they occur in superior arm most frequently. More rarely the lower-arm type has occurred; very rarely total palsy is reported (10 cases). Now and then a case of individual muscle paralysis has been reported in obstetric palsy.

ETIOLOGY (A. S. TAYLOR).

The predisposing factors in the production of this lesion are such disproportion between the size of the child and the maternal pelvis as would delay the easy progress of the labor, and especially as would hinder the rotation and descent of the shoulders after descent and extrusion of the head, rigidity of the maternal soft parts, maternal exhaustion, and any other cause which will delay labor and therefore lower the vitality of the fetal tissues.

Apparently, from the cases on record, transverse and face presentations have not been responsible for the production of many of these cases.

They occur with about equal frequency in vertex and breech presentations. (Schümacher, 93 cases: 50 vertex, 43 breech.)

Brachial birth palsy occurs in about one in two thousand labors (Duvall and Gullian, 1901). If one admits that laceration birth palsy occurs in but one-half of all the cases, the ratio stands one in four thousand for that type of palsy which is the one under consideration here.

It is erroneous to suppose that laceration birth palsy occurs only in mismanaged labor; it may easily result in highly skilled hands, as shown by its occurrence at the birth of Emperor William of Germany, on which occasion Sir William Jenner was the accoucheur.

It is manifestly important to determine the usual site, extent, and nature of the lesion, as well as the etiological factors concerned as exciting causes of the lesion.

Those factors commonly mentioned are:

I. Backward pressure on the nerves by the clavicle, (*a*) on transverse processes of the vertebræ; (*b*) on first rib.

II. Hyperextension of the arms in breech cases.

III. Pressure of forceps.

IV. Tension on the nerve roots.

It is noticeable that, while so many different causative factors are cited, the results are invariably the same in type, though differing in degree. This strongly suggests that one fundamental type of nerve injury underlies all the cases.

To determine which of these factors is the essential one, twenty dissections were made upon ten infants within three to ten days after death. The skin, fasciæ, and fat were removed from the lower part of the posterior cervical triangle to fully expose the clavicle and

nerve roots. Also the pectoralis major and deltoid muscles, with skin, fat, and fascia were removed below the clavicle. This gave a free exposure of the brachial plexus, vessels, and bony structures.

The following anatomical facts were noted:

(*a*) The clavicle is small, has a smooth, rounded, posterior surface, is so curved as to fit around the side of the neck without localized pressure, is somewhat flexible, and the ligaments of its articulations at both ends are so loose as to allow free play of the bone in any direction.

(*b*) The first rib is small, flat, and very flexible.

(*c*) The nerve roots come out between the transverse processes of the cervical vertebræ, of which the anterior tubercles lie well forward of the nerve roots.

(*d*) The nerve roots lie between the scalenus anticus in front and scalenus medius muscle behind, which form a protecting muscle cushion.

(*e*) There is a thick layer of fat and fascia behind and below the clavicle.

(*f*) The clavicle lies well below the usual site of lesion in brachial birth palsy (*i. e.*, fifth and sixth roots) and can be pushed up to that site only with some difficulty.

(*g*) When the clavicle is pushed directly backward with the arm at the side, or when it presses backward during hyperextension of the arm, (1) the nerves slide outward away from the lines of pressure; (2) the nerves are pressed upon so little that they slide up and down behind the clavicle with the greatest freedom when traction is applied to them.

(*h*) In hyperextension of the arm the clavicle rotates backward and slightly downward. It does not rise to the level of the lesion commonly found in brachial birth palsy. The nerves are not rendered tense by this manipulation.

Reasoning from these data, backward pressure by the clavicle is not a factor, inasmuch as the bone does not reach to the level of the usual lesion; the anterior tubercles of the transverse processes prevent it from pressing the roots at their exit from the spine; the first rib is so flexible as to give scarcely any resistance to pressure; the nerves are surrounded and protected by muscle and fat cushions, and the clavicle is so curved, so smooth posteriorly, and so loosely attached at either end that when it is displaced strongly backward the nerves can still be easily moved either sideways or up and down, showing an absence both of pressure and tension.

If, however, in exceptional cases, the nerve should be compressed by the clavicle, the result would be a narrow transverse lesion of moderate degree which would always go on to spontaneous regeneration. Moreover, if the lesion were the result of compression between the clavicle and first rib, the lower roots of the plexus should show most damage because they are so placed as to sustain the

major part of such pressure. In all cases, however, the fith and sixth roots are the ones most damaged.

Hyperextension is not a factor, inasmuch as it neither causes pressure by the clavicle nor causes the nerves to be stretched tightly around the clavicle.

Forceps cannot be a factor of moment because the blades will not reach as low as the site of the lesion when traction is applied. Moreover, if they could cause a lesion it would be a narrow transverse one, which would recover spontaneously as do the facial palsies resulting from forceps pressure.

Another fact against any of these kinds of pressure being the cause of the lesion is that in the seven cases thus far operated upon 1 to 2 cm. of the length of the nerve were involved, whereas any lesion the result of pressure from the clavicle or forceps would not involve more than 3 to 7 mm. of the nerve length.

There remains the consideration of tension as the causative factor. Some authors say it is *a* factor; some that it is *one* of the *chief* factors.[1] In the dissected specimens above mentioned the *only* factor which caused damage to the nerve roots was tension, and tension sufficiently great caused lesions in the same situation and of like nature to those found in the seven cases operated upon. Therefore, tension is the only factor concerned in the production of persistent (laceration) brachial birth palsy of the Erb type. When tension is present certain other factors may increase the amount of damage to the nerves, as we shall see later.[2]

The dissected infants were placed in all the attitudes incident to delivery in either vertex or breech presentations, and it was found that only one thing caused stretching of the nerves, namely, increase in the distance from the head and neck to the shoulder. Also, in six living infants from three to ten days old, it was found that pushing the head and shoulders apart causes the upper nerves of the plexus to stand out under the finger like fiddle-strings, while in any other position they were either relaxed or not palpable at all.

In vertex presentations this attitude occurs when the shoulder is obstructed either at the brim of the pelvis or by the symphisis pubis, and the head is pulled upon by the accoucheur with instruments or hands. If rotation or oscillation of the head and neck be added the strain is increased and more damage is apt to occur. A

[1] The connective-tissue overgrowth found at autopsy in some of these palsy cases was described in one case as a "tumor growth" (Eversmann); in another as "neuroma" (Parry); and in a third was considered as the result of a "maternal impression" (Bachrach). A few observers still believe the essential cause to be a form of congenital dislocation of the shoulder from which they deduce a plan of manipulative replacement therapeutics.

[2] H. L. Taylor reports that some form or degree of palsy occurs in about 25 per cent. of reduction of congenital hip dislocations by Lorenz's method. The lesion in such is undoubtedly of a like character to that shown here in obstetrical palsy. Probably the irreparable lesion of the sciatic nerve which he reports is comparable to the very severest lesions of brachial birth palsy in which the nerve and sheath have suffered complete loss of continuity. It is next necessary to determine how the tension is brought about.

very few cases are known where, after a normal vertex labor with neither instrumental nor manual traction, a temporary birth palsy has occurred.

In breech presentations the lesion occurs in the delivery of the after-coming head. The fingers hooked over the back of the neck pull both shoulders down and away from the head and neck. Here again rotation or oscillation increases the strain on the nerve roots. Moreover, the tips of the fingers, hooked over the shoulders, lie upon the stretched nerves and add a lateral strain to the tension they already suffer from.

It is evident then that any form of traction which pulls the head and neck away from the shoulder is the exciting cause of the lesion. As a natural corollary the lesion may occur with any "presentation."

It was most interesting to note the nerve area involved and the sequence of events in the production of the lesion on the cadaver.

It was invariably the case that the fifth root gave way first, then the sixth, and so on down the plexus in regular order if the force used was sufficient. From the lay of the plexus it is perfectly obvious that the fifth root must bear most of the strain until it yields, then the sixth, and so on downward.

This same fact is borne out clinically, inasmuch as the mildest cases show only paralysis of muscles belonging to the fifth root and progress from that to cases involving rupture of the entire plexus.

The nerves do not give way in a limited transverse section, but fray out over a considerable linear area, just as does a rope when it is overstrained. This fact accounts perfectly for the pathological pictures found in our seven cases at the time of operation.

The location of the lesion is important and will be more easily understood if it is remembered that the fifth and sixth roots fuse about 1.5 to 2 cm. (in infants) from their spinal exits. Their junction is from 0.5 to 1 cm. long, and from its distal end pass two nerves to form part of the outer and posterior cords of the plexus respectively. For convenience sake, these may still be called the fifth and sixth nerves, and in locating the lesion the terms "above," "at," and "below the junction" will be used.

In the twenty experiments cited the lesion was produced:

	Fifth nerve.	Sixth nerve.
Above junction	16 = 80 per cent.	17 = 85 per cent.
At junction	2 = 10 "	2 = 10 "
Below junction	2 = 10 "	1 = 5 "

In the case of the sixth nerve, 7 (35 per cent.) of the ruptures consisted in avulsion of its root, including the root ganglion from the spinal cord.

Great force must be employed to cause the lesion, which accounts for the relative infrequency of its occurrence.

Of the 7 operative cases, 1 (Fig. 6, Case I.) involved rupture of the entire plexus. Of the remaining 6, 5 (83 per cent.) showed the

maximum damage above the junction and 1 (17 per cent.) below it, about the same percentages as in the experimental cases. In all the cases the cicatricial sequelæ of the injury involved the "junction."

In 1 case (Fig. 6, Case VII.) the fifth root was torn across above the junction, the ends separated about 1 cm., and bound down by connective tissue.

In 1 case (Fig. 8, Case III.) the fifth root was torn across just below the junction, and the distal end displaced inward and downward about 2.5 cm. to the front of the scalenus anticus muscle, where it was adherent.

There was no case of avulsion of the sixth root.

It will be seen in these cases that the maximum damage is to the fifth root.

A nerve of special importance which is always involved is the suprascapular. It is a small nerve arising from the distal and outer aspect of the junction of the fifth and sixth roots and supplies the external rotator muscles of the shoulder.

PATHOLOGY (T. P. PROUT).

To properly appreciate the lesion in these cases the etiological importance of the overstretching process in its production at birth must be borne in mind. Furthermore, a study of the lesion should demonstrate not only the characteristics of the degenerated areas, but the reasons for the failure of complete regeneration following the accident. The severed ends are in apposition in the vast majority of instances; when such condition is brought about surgically, union, regeneration, and recovery of function occur without incident. Why are these cases exceptional in this particular?

In this connection several points must be borne in mind:

1. The force producing the lesion is variable; the lesion therefore varies in extent with the variation in the extent and character of the force producing it, as has been pointed out. In the milder (most usual) cases only the fifth and sixth cervical roots suffer (Case VI.), while the severer cases present a literal severing of the roots of the plexus, varying in number from one (Case VII.) to three or even more (Case I.).

2. The nature of the force producing the lesion is such that the nerve roots are *pulled* apart rather than severed clean at a given point. The lesion, therefore, is incomplete at any given cross-section of the nerve bundle and involves different fibres at different levels.

3. The nerve bundle is surrounded by a dense sheath of connective tissue, the perineurium, which supports it and the vessels supplying it. In any lesion of the nerve roots resulting from an overstretching process this supporting sheath must first give way. In these cases it is torn asunder and the arterioles belonging to it and supported by it are ruptured. A hemorrhage into and beneath the

perineural sheath and infiltrating the strands of nerve fibres and the meshes of the epineurium results. In the recent state a small hæmatoma infiltrating these structures forms at the point of rupture, as shown in the accompanying schematic diagram (Fig. 1).

FIG. 1

Schematic representation of a rupture of the perineural sheath and hemorrhage into its substance and into the meshes of the epineurium resulting in the formation of a hæmatoma: *a*, epineurium; *b*, perineurium; *c*, nerve bundles; *d*, hemorrhage.

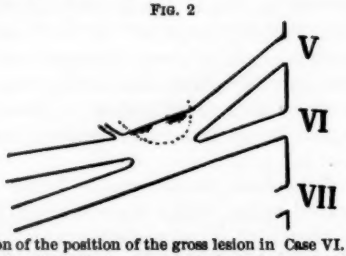
4. This accident occurs at birth, a period of life when the vascular and nervous structures are susceptible of injury of a far-reaching character.

Among these various points only those of immediate import in the production of the lesion interest us here.

Of the immediate lesion little has been written. Strange to say, it is scarcely mentioned in medical literature, there being no adequate description of the gross lesion, and none whatever of its microscopic appearances. The gross appearances vary much with the duration and severity of the case.

In all of the operative cases here detailed the deep cervical fascia was invariably found greatly thickened, especially over the plexus, and irregularly adherent to the nerve roots. The thickening of the fascia was of an irregular and variable character, depending upon the severity of the case.

FIG. 2

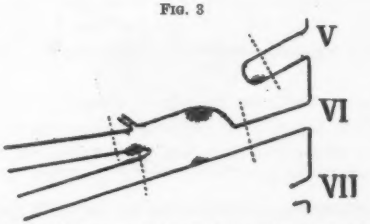


Schematic representation of the position of the gross lesion in Case VI. The dotted line represents the area excised, and the dark areas within it the lesions in the perineural sheath.

In cases of the milder grade, such as Case VI. for example, the outward appearance of the nerve strand at the point of the lesion may be normal, but in this particular instance a distinct nodular mass could be felt in the upper and outer aspect of the nerve strand (Fig. 2).

In this instance also the duration of the lesion was such that the *gross* abnormality was reduced to a minimum. In Case VII., on the other hand, the lesion had existed but eleven months and was, therefore, relatively fresh, and may be taken as presenting some of the more characteristic points of a relatively recent lesion of the severer grade. In this instance there was a severance of the fifth cervical root just before its junction with the sixth root (Fig. 3).

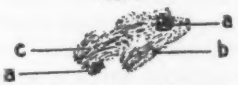
FIG. 3



Schematic representation of the gross lesion and its extent in Case VII. The dotted lines show the area excised. The actual size and shape of the larger piece is shown in Fig. 4. The dark spots represent the positions occupied by the various hæmatomata resulting from the rupture of the perineural sheath and hemorrhage into its substance and the meshes of the epineurium.

In this case the severed ends were more or less rounded and embedded in a hæmatomatous mass, as shown in Fig. 4, which is a drawing of a section through the entire length of the larger piece excised in this case. The larger of these hæmatomata is represented at *a* in the figure, while there are smaller ones at *b* and *c*, all of which represented nodular masses in the gross specimen quite distinct in each instance.

FIG. 4



Actual size of piece excised in Case VII. *a*, small hæmatoma in the perineural sheath and epineurium; *b*, smaller hemorrhage with marked infiltration beneath the perineural sheath; *c*, strands of nerve fibres.

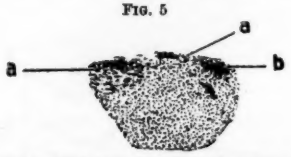
The recognition of the importance of the rupture of the perineural sheath and the resulting hemorrhage into its substance and the substance of the nerve bundle is not mentioned in works upon this subject.

These are points of great importance, since they are *the* determining factors in the production of the ultimate lesion. Were it not for these, we might reasonably expect a recovery more or less complete in all cases except those few presenting an actual rupture of the roots of the plexus. These factors, however, determine the ultimate extent and final character of the lesion.

The extent of the lesion varies greatly with the severity of the

case. Fig. 2 presents in schematic form the extent of the lesion in one of the milder cases. In this instance the lesion occurred at the customary site at the junction of the fifth and sixth cervical roots. The dark areas in the figure represent the extent of the lesion in the nerve sheath as developed in a series of sections lengthwise through the piece excised. A drawing of the entire section slightly magnified, showing the distribution of the lesion, follows in Fig. 5.

FIG. 5



Longitudinal section of piece excised from Case VI., magnified 2: *a*, perineural sheath; *b*, pigment deposits and connective tissue infiltrating the substance of the nerve bundle.

In Case VII. there was a severance of the fifth root. The extent of the lesion and the area excised is shown in Fig. 3. In this instance the lesion was comparatively recent and fairly well organized hæmatomata were present in the nerve sheath, as already mentioned. Fig. 4 shows the larger of the excised pieces, actual size. The position of the various hæmatomata is also indicated. In Case III. the lesion was still more decided, the fifth root being severed below the junction with the sixth and the excised mass (junction of the fifth and sixth roots) being involved in a ruin more or less complete. In this instance the cells of the neurilemma which represent the remains of the nerve fibres are thrown into folds, one of which is shown at *A* in Fig. 12, 3. The fibres *A* come to an abrupt ending in a connective-tissue mass, *C*, an old organized hemorrhage, with some masses of pigment still remaining in the interior.

The types of lesions depicted in these three cases represent fairly well the varying grades usually met with.

A conception of the ultimate lesion is to be obtained only by a study of the sequence of events in the nerve trunk following the primary lesion described and depicted above.

Organization of the blood clot or hæmatomatous mass and a union of the severed perineural sheath take place before any attempt at regeneration of the nerve fibre itself. The new connective tissue formed in this process contracts upon the nerve bundles and prevents regeneration of the nerve fibres. If the lesion in the perineural sheath is considerable, resulting in the formation of hæmatomata at the points of rupture, as in Case VII., the effect of the organization of the blood-clot will be such as to prevent the regeneration of a relatively large number of nerve fibres in the nerve bundle. Such points of compression are illustrated in Fig. 11, Cases II. and V. In the latter instance the lesion was too recent to show the disfiguring effect of its contraction upon the nerve fibres.

In all of the cases thus far examined the evidences of an old hemorrhage still persist in the tissues (Fig. 12, 1, *B*, and 4, *A*). In some of these areas the section presented a fairly large vessel in the immediate neighborhood of the old hemorrhage which may easily have figured in the production of the original lesion.

A further possibility following the rupture of the perineural sheath is shown in Fig. 12, 2 and 4. In the first a dense band of connective tissue, *A*, crosses the nerve bundle and strangles it. In the latter there has been a buckling inward of the perineural sheath as shown at *A*, and also in Fig. 13, 2, *A*. This buckle in the sheath effectually strangulates the nerve bundle and has prevented its regeneration.

As regards the behavior of the nerve fibre caught in lesions of this character, its appearance varies very much with the extent and duration of the lesion at a given point. Fig. 13, 1 and 3, present areas of extreme compression. In the unstained areas nothing remains except the cells of the neurilemma, together with some old fragments of the myelin sheath. 3 presents in particular the results of an incomplete compression of the nerve trunk.

From a study of these sections it is evident that the lesion in these cases is not confined to any one given level, but even in the milder grades of palsy extends over quite a wide area.

Some special features in connection with particular cases deserve notice:

In Case VI. there were two lesions in the perineural sheath separated by a short distance (shown in text, Fig. 5) and varying greatly in consistency. The mass at *b* was a hard nodule quite readily felt in the gross specimen; while the lesion at b^1 was more diffuse and the tissue of looser arrangement. Both of these areas contain some pigment deposits, the remains of an old hemorrhage which originally involved the nerve trunk diffusely and has since undergone organization, choking the involved nerve fibres in a contracting connective-tissue mass.

Case VII. belonged to the severer type and differed from the other cases in which the tissue has thus far been examined in being of comparatively recent origin. In the area excised there were two hæmatomata, each about the size of a small pea, which had developed in the substance of the epineurium (Fig. 4, *a*).

The sequence of events in the production of the laceration brachial birth palsies may be summarized as follows:

The lesions produced are (*a*) immediate and (*b*) remote.

The immediate lesion consists in the tearing of the perineural sheath surrounding and supporting the nerve trunk and the incidental rupture of the bloodvessels belonging to it. Hemorrhage occurs into and beneath the perineural sheath, and in many instances into the substance of the epineurium. There is, furthermore, a severance of the nerve strands more or less complete, depending on the severity of the case.

The remote lesion is brought about and its extent determined by (*a*) the healing of the rent in the perineural sheath; (*b*) the size of the blood clot and its ultimate organization; (*c*) the ultimate contraction of the cicatrix upon the nerve strand, which not only prevents its regeneration, but determines a neuritis in certain of those, not severed, upon which it may chance to impinge.[1]

SYMPTOMATOLOGY AND DIAGNOSIS (L. P. CLARK).

The "laceration palsy" dealt with here is so characteristic in type that in severe cases its clinical recognition, from the attitude alone, is easy; thus the arm hangs powerless by the side, and cannot be abducted at the shoulder because of the palsy of the deltoid and supraspinatus muscles; the forearm is extended, and cannot be flexed on account of paralysis of the biceps, brachialis anticus, and supinator longus; the hand is in extreme pronation, caused by palsy of the supinator brevis and biceps, and the entire arm is so rotated inward that the palm of the hand may look backward and outward. The humerus is markedly rotated inward as a result of paralysis of the supraspinatus and infraspinatus and teres minor muscles.

Where laceration of the nerve roots has occurred there is present a factor of very great clinical importance not heretofore recognized and of fundamental significance in the early treatment and prognosis of the condition. When an infant remains peevish and fretful for a considerable period after this accident at birth, and when handling of the extremity greatly increases the pain and irritability, there is present a traumatic neuritis aggravated by pressure incident to the organization of the blood clot and repair of the rent in the perineural sheath.

Cases that do not present this symptom will show a more or less complete spontaneous recovery; while in those cases presenting these signs of a neuritis a considerable palsy will follow, depending on the severity of the lesion.

In severe cases after several months the internal rotators, especially the pectoralis major, become markedly contractured, further increasing the deformity of the arm at the shoulder-joint, and this, combined with the paralysis of the external rotators, may lead to more or less posterior displacement of the head of the humerus (as in Case III.). There are present also some contracture at the elbow preventing full extension and more or less flexion contracture and ulnar adduction at the wrist. These attitudes are well seen in all the case pictures. The limits of motion are well shown in Figs. 7 and 8.

[1] In some cases of extreme laceration brachial birth palsy, both congenital torticollis and spasmodic wryneck are also present. From the nature of laceration birth palsy and its pathogeny it is probable that the lesion in the above conditions is a nerve injury resulting from compression or stretching, and not a lesion of the muscles as Stromeyer contends. The spasmodic character of these affections can be explained on the basis of incomplete nerve regeneration, as is true of the spasmodic conditions in chronic facial palsy.

The clinical picture of laceration brachial birth palsy (chronic) is well shown in Figs. 7 and 8, 1. The muscles affected do not belong to the distribution of any single nerve trunk of the arm involved. Erb indicated the exact point at which the lesion could be demonstrated, and this point has been called by his name. The site is about 2 cm. outside the sternomastoid and the same distance above the clavicle, as shown in Fig. 8, 4. Erb demonstrated that by means of electric stimulation at this point one may evoke contraction in all the muscles found affected in this type of paralysis. This point corresponds to the junction of the anterior divisions of the fifth and sixth cervical nerve roots. It has been shown by Erb experimentally, and later by others through clinical study, that the motor fibres to the deltoid, biceps, brachialis anticus, supinator longus, and sometimes supinator brevis muscles run in the junction of the anterior divisions of the fifth and sixth nerve. The palsy resulting from an injury of the nerves at this point is obvious. Orientation of the site of the lesion may be facilitated by referring to the semidiagrammatic scheme of the cervical nerve roots and the formation of the brachial plexus by their union (Fig. 9).

In laceration birth palsy the fifth and sixth cervical nerve roots suffer first. Frequently the seventh and occasionally the eighth cervical and first dorsal nerve roots are involved, thus constituting a lesion of the entire plexus as shown clinically in Fig. 6, 1. In not a few cases of the laceration palsy the entire arm is paralyzed, but for a time only. In such, the milder degree of lesion in the lower cervical nerve roots is recovered from in the course of a few months. Sufficient data are not yet at hand to declare the exact nature of the stretching lesion in these lower cervical nerve roots which recover in part. It seems probable, however, that temporary palsy results here in consequence of simple stretching of the nerve strands without causing rupture of the bloodvessels or the perineural sheath itself. Certainly a lesion of the severe type hereafter to be described could not be present in any marked degree and spontaneous recovery take place.

The sensory changes in brachial birth palsy are very slight and do not aid in the diagnosis. The overlapping of the segmental cord supply to the skin in the affected parts may account for the absence of sensory changes. Although careful tests were made, no detectable sensory changes were present in this series of cases either before or after operation.

The amount of atrophy is moderate, but the developmental defect which is noticeable after a time may be slight or severe, and apparently it is more dependent upon the extent of the brachial lesion than upon any degree of local damage to the plexus nerve. A case now under observation shows extreme dwarfing of the entire upper limb, although all the muscle that is now present seems of good quality, and there is but slight limitation of the motions of the ex-

tremity. The diagnosis is based on the history of traction used at delivery, followed immediately by paralysis of the upper extremity, with the assumption of the attitude and characteristics mentioned just previously in the symptomatology. The degree and extent of lesion in any birth palsy case cannot be determined immediately after its occurrence. There is no definite test to determine whether the nerves have been divided, simply compressed, or lacerated and contused by overstretching. Electric tests are only possible in the palsied child when it is placed under anæsthesia, and even then the results of such tests are contradictory and misleading, a fact early recognized (Stransky, Soltman, Westphal). The diagnosis of the range of nerve injury may be made approximately by analysis of the resulting muscle palsy, the atrophy and extent of growth defect. The electric reactions of the muscle (R. D. of paralyzed muscle) also assist in this particular. In the course of a few months one may detect by palpation a thickening and induration of the nerve roots at Erb's point. By palpation a fairly accurate knowledge of the state of the whole plexus may be obtained in those children who are not fat and who assist the examiner by moderately shrugging the shoulders. By practice one soon gains expertness in this diagnostic aid. The following table embraces the most recent data of the neuromuscular anatomy of the parts affected in birth palsy, and aids much in determining the distribution and degree of lesion present in a given case. It should be used in connection with Fig. 9.

This table shows the immediate nerve supply, plexus division, and nerve-root origin of the muscles of the shoulder, arm, forearm, and hand.

MUSCLES MOVING SCAPULA AND ARM.

Muscle.	Immediate nerve supply. *Plexus Division.*	Root origin.
Trapezius.	Spinal accessory.	C. II. to VI.
Rhomboideus.	Spinal accessory.	C. IV. and V.
Levator angulæ scapulæ.	Spinal accessory.	C. III., IV., V.
Serratus magnus.	Posterior thoracic.	C. V., VI.
Deltoid.	Circumflex (posterior cord).	C. IV., V.
Supraspinati and infraspinati.	Suprascapular.	C. IV., V.
Teres minor.	Circumflex.	C. V.
Subscapularis.	Short subscapular.	C. V., VI.
Latissimus dorsi.	Long subscapular (posterior cord).	C. VII.
Pectoralis major.	Anterior thoracics (outer and inner cords).	C. V. and VI.
Teres major.	Suprascapular (posterior cord).	C. VI., VII.

MUSCLES MOVING FOREARM.

Triceps.	Musculospiral (posterior cord).	C. VI., VII.
Brachialis anticus.	Musculospiral and musculocutaneous (outer and posterior cord).	C. V., VI., VII.
Biceps.	Musculocutaneous (outer and posterior cord).	C. V., VI., VII.
Supinator longus.	Musculospiral (posterior cord).	C. V., VI., VII.
Supinator brevis.	Musculospiral (posterior cord).	C. V.
Pronators (teres and quad.).	Median (outer and inner cord).	C. VI., VII.

MUSCLES MOVING THE HAND.

	Flexors of Wrist.	
Flexor carpi uln.	Ulnar (inner cord).	C. VIII., D. I.
Flexor carpi rad.	Median (inner and outer cord).	C. VII., VIII.
	Extensors of Wrist.	
Extensor carpi rad. longior. Extensor carpi rad. brevior. Extensor carpi ulnaris	Musculospiral.	C. VI., VII.
	Extensors of Fingers.	
Extensor communis digitorum.	Musculospiral.	C. VI., VII.
Extensor indicis.	Musculospiral.	C. VI., VII.
Extensor minimi digiti.	Musculospiral.	C. VI., VII.
	Flexors of Fingers.	
Flexor sublimis.	Median (inner and outer cord).	C. VII., VIII.
Flexor profundus.	Median and ulnar (inner, outer, and posterior cord).	C. VII., VIII. D. I.
Interossei and lumbricales.	Ulnar (inner cord).	C. VIII., D. I.
Two outer lumbricales.	Median (inner and outer cord).	C. VIII., D. I.

PROGNOSIS (L. P. CLARK).

The prognosis in birth palsy depends upon the (*a*) nature and degree of the nerve injury; (*b*) the treatment.

(*a*) In the mild cases where possibly compression alone (rare) or at least only the slightest grade of laceration occurs, spontaneous and fairly prompt recovery may be expected (three to nine months). However, even in these cases there may be a persistent atrophy and retarded development through life of a part or the whole of the extremity, although the range of motions may be little or not at all restricted.

In more decided lacerations of the nerves the outcome depends on how much the cicatricial contractions in and about the nerves (see Pathology) obstruct the anatomical and functional continuity of their fibres. There may, then, result any condition from nearly complete recovery to the total loss of function of a part or the whole of the extremity.

Inasmuch as it is impossible, just after the injury (diagnosis), to determine the exact distribution and degree of the lesion, only time (six to twelve months) will permit a fairly accurate prognosis to be made.

In those cases presenting definite "traumatic neuritis" of the nerve roots (symptomatology) the prognosis is relatively bad from the outset, and it may be positively stated that there will be some degree of permanent damage to the extremity, although here again only time can determine its exact extent. In many cases the lesion consists of several foci scattered through the nerves at the site of injury. Therefore the prognosis is influenced by the distribution as well as the degree of the lesion.

The deep cervical fascia overlying the nerve roots is injured in all the severer cases, and by its cicatrization and adhesions to the

nerve roots (noted in all 7 operated cases) interferes with their regeneration.

(*b*) Proper treatment greatly influences the prognosis even in those cases which will suffer from permanent nerve damage, for, as stated in the symptomatology, there is always some recovery in the nerves which have been only moderately damaged about the periphery of the maximum laceration. During the recovery of these nerves and muscles, unless proper treatment (see Treatment later) is carried out, there occur contractures of muscles and ligaments, with resulting deformities of the joints which very greatly limit the functional value of such muscles as regain power.

Only 26 per cent. of all forms of brachial birth palsy recover (Bruns).

It has been stated (Comby) that cases which occur in head presentation have much the better chance of recovery. While this statement is borne out by statistics, yet one can understand that as less stretching force is apt to be applied in vertex labor, these cases would naturally show less serious lesions.

TREATMENT (CLARK AND TAYLOR).

(*a*) *Prophylactic.* The necessity of prophylaxis is obvious and it can easily be deduced from a consideration of the "etiology."

(*b*) *Palliative.* Inasmuch as the degree of spontaneous recovery which will occur in any given case cannot be determined until after many months, treatment should be directed to maintaining the nutrition of the muscles, to preventing contractures and resulting deformities, and to assisting the spontaneous repair of the damaged nerves by means of massage, passive motion, douches (hot and cold), electricity, and such apparatus (if necessary) as will overcome a tendency to deformity.

It must be remembered, however, that in the cases showing marked evidences of traumatic neuritis, complete immobilization is demanded until the inflammatory reaction has subsided (often two to four months). During this period means should be taken in the immobilization to hold the extremity in normal position (*i. e.*, overcome the internal rotation) and so prevent contracture of the pectoralis major muscle and the anterior ligaments in that attitude. The attempt to use the active measures above detailed tortures the child and aggravates the inflammatory reaction. When the neuritis has subsided the active measures may be employed.

Electricity not only helps keep the muscles in good condition, but also aids materially in the nerve regeneration by stimulating "neurofibrillation" in the regenerated homogeneous axis cylinders (Bethe).

(*c*) *Radical.* The length of time through which palliative treatment should be continued is still a matter for discussion. If, by the

end of the year, complete recovery has not occurred, a prognosis of permanent lesion may be definitely made, although some slight improvement occurs after this time in certain cases.

Kennedy counsels operation at the end of three months, but that would seem too early, inasmuch as, at that time, none of the spontaneous regeneration, which occurs to some degree in nearly all cases, would have had time to show itself and to permit of a more accurate diagnosis of the distribution and degree of the lesion.

If the palliative treatment be systematically employed there would seem to be no disadvantage in waiting a year before proceeding to radical measures, although in certain selected cases a shorter time limit might be chosen (where electric responses show no tendency toward spontaneous improvement after four to six months).

The advantages of waiting lie in the increased size of the operative field, the more definite localization of the lesion, and the lessened danger to life from shock and hemorrhage.

The clinical demonstration that there is to be a permanent lesion in any given case means that there must be a cicatricial lesion preventing complete nerve regeneration, as has been proven in the consideration of the pathology of the condition.

Here then, as in other traumatic lesions of the peripheral nervous system, the only rational treatment lies in the excision of the damaged tissue, with following nerve suture.

There have been but few secondary operations upon the brachial plexus. Excision of the lesion in a case was advised several years ago (Starr), but owing to the extent of the lesion found the operation was abandoned.

In 1900 Kennedy operated upon a case in which the brachial plexus was involved in a cicatrix causing total palsy of the arm. An operation for the relief of the nerve from adhesions resulted in a practically complete recovery fourteen months after operation. The same author reported operations upon three additional cases of birth palsy in February, 1903. Case I. made a nearly complete recovery in nine months after operation. Case II. five months after operation showed improved electric reaction, but no return of voluntary power. Case III. only three months after operation showed returning faradic response. He recently reported three additional cases, but the time was too short for results to have appeared.

OPERATIVE TECHNIQUE (A. S. TAYLOR).

The patient is anæsthetized and brought to the table with the field prepared for operation. A firm cushion is placed beneath the shoulders, the neck is moderately extended, and the head held toward the sound shoulder. This position gives a good exposure of the operative field.

The incision passes from the posterior border of the sternomastoid

muscle, at the junction of its middle and lower thirds, downward and outward to the clavicle at the junction of its middle and outer thirds. After the skin, platysma, and deep fascia are divided, the omohyoid muscle is exposed near the clavicle, and lying beneath it are the suprascapular vessels. These structures may be retracted downward, or, if the case requires the extra room, the omohyoid first and then the vessels, between double ligatures, may be divided. The transversalis colli vessels are encountered a little below the middle of the wound and divided between double ligatures.

The dissection is rapidly carried through the fat layer to the deep cervical fascia covering the brachial plexus. In all the cases this fascia was thickened at the site of lesion of the nerve trunks. This fascia is divided in the direction of the original incision, is dissected away from the nerve trunks to expose them freely (Fig. 10, 1). The damaged portions of nerve are usually noticeably thickened and of greater density than the normal nerves. The extent and distribution of the paralysis, determined before operation, gives the clue as to which roots are at fault. Usually the junction of the fifth and sixth roots is the site of maximum damage. The thickened indurated areas are determined by palpation and are excised by means of a sharp scalpel. Scissors should never be used for this purpose as they pinch the nerve ends so much as to seriously interfere with their later regeneration.

The nerve ends are brought into apposition by lateral sutures of fine silk involving the nerve sheaths only, while the neck and shoulder are approximated to prevent tension on the sutures. Cargile membrane is wrapped about the anastomosis to prevent connective-tissue ingrowth. The omohyoid muscle, if it has been divided, is sutured with catgut. The platysma and skin are sutured with silk. A firm sterile dressing is applied, and the bandage is made to approximate the shoulder to the head (Fig. 6, Case VI., 2). This position must be maintained for from two to three weeks to permit of firm union between the nerve ends. In the first five cases it was very difficult to maintain this position by bandages, especially during the struggling incidental to recovery from the anæsthetic. It must be remembered that a single moment's separation of head and shoulder after the nerve sutures are tied will spoil the operation. Preceding Case VI. a plaster splint was moulded to the head and paralyzed extremity so as to maintain them in the desired relative position. After hardening it was removed and lined with soft material. At the operation, as soon as the nerve sutures were tied, the cast was slipped on and the skin wound sutured without danger of tension on the nerves (Fig. 6, Case VI., 2). The child could readily be lifted about later without danger to the suture. A neater splint was designed later, with buckles and webbing straps to eliminate the necessity for bandages (Fig. 6, Case VII., 2).

It will be noticed that (Fig. 10, 2) (*a*) the tissues to be excised lie

in close proximity to the phrenic nerve and internal jugular vein, and to the junction of the cervical sympathetic communications with the spinal nerve root. (*b*) The suprascapular nerve comes off from the junction of the fifth and sixth cervical nerve roots, which, as already stated, is usually the site of maximum damage. This nerve is very small in children, but it should be sutured with the greatest care to proximal nerve stump, since it innervates the external rotators of the shoulder, the paralysis of which permits the posterior dislocation of the shoulder often seen in the older cases.

In cases where the lesion is more extensive, especially where it lies beneath the clavicle, a wider exposure is necessary (Fig. 10, 2). It is obtained as follows: The first skin incision is carried downward along the line between the pectoralis major and deltoid muscles, which are then separated. The clavicle is divided in the line of the skin incision by a Gigli saw; the subclavius muscle is divided as are the omohyoid muscle and suprascapular vessels if they have been spared in the earlier steps of the operation. When the outer fragment of the clavicle and the shoulder are retracted outward the entire brachial plexus, with more or less fat in front of it, is exposed down to the upper margin of the pectoralis minor muscle. (If this muscle is divided a still wider exposure is given, but this is seldom required.)

When the nerve suture is completed the divided muscles are repaired, the clavicle is sutured with chromic gut, and the skin closed with silk as before.

This operation, because of its length, the loss of blood, and the shock involved, is applicable only to older children.

Where the lesion involves the whole plexus it would be wise to do only a portion of the repair work at the first operation and complete the repair at a later operation, thus avoiding too long and exhausting a procedure at one time. (See Case I.)

After-treatment. When the immobilization is removed, three weeks after operation, the measures indicated in Palliative Treatment are systematically employed. As soon as evidence of returning voluntary power appears in the paralyzed muscles, attention should be given to encouraging voluntary motions in all directions, thus educating the motor centres of the brain to control muscles that were up to that time beyond their influence. Improvement, though slow at first, will continue for a number of years until more or less complete recovery occurs.

A definite prognosis as to the amount of recovery that will follow operation cannot be given until several months have elapsed.[1]

[1] In the older cases where contracture deformities are already present the procedures advocated by Whitman (New York Surgical Society, March, 1905) are of great assistance in regaining the usefulness of the limb. The method consists in forcible reduction of the deformities under ether in several sittings, and the maintenance of improvement, so gained by forcible passive motion in the interval, based on the principle of Lorenz's reduction of congenital dislocation of the hip.

HISTORY OF CASES (A. S. TAYLOR).

During the past two years seven cases have been operated upon by the method detailed. Their histories are as follows:

CASE I.—I. H., aged twenty-five months. Patient is a girl who presented complete paralysis of the left arm, as shown in Fig. 6, Case I., 1. A complete lesion of the entire brachial plexus was diagnosticated. She was a first child, vertex presentation. Labor was prolonged and difficult. Instruments were used and strong traction exerted on the head. Complete paralysis of the left arm was noted a few days after birth. No spontaneous improvement resulted during the first two months after birth. After four months, however, slight flexion of the fingers and wrist was possible. In every other way the child was perfectly healthy. There was R. D. in all the muscles of the upper extremity. Contractures produced the deformity shown in the case. An indurated area corresponding to the site of the sixth, seventh, and eighth cervical nerves were found on palpation.

Operation May 18, 1903; chloroform. The more extensive exposure as described above (Fig. 10, 2) was made in this case. The region of the normal brachial plexes was occupied by dense fibrous tissue, in which it was difficult to make out any nerve trunk. After some dissection the proximal ends of the nerves were identified, and the nerves were divided near the outer border of the scalenus anticus muscle. Distally, the nerves were identified and the division made just beyond the cicatricial area, which was in all about 2.5 cm. wide (Fig. 11, Case I.). The nerves were sutured to the proximal stumps with silk, the clavicle was united with chromic gut, Cargile membrane was wrapped around the anastomoses. The wound was closed without drainage. The operation lasted somewhat over one and one-half hours; a moderate amount of blood was lost. The child was returned to bed in fair condition about 3 P.M. Five hours later the temperature was 106°; pulse, 180; respirations, 68. In spite of bathing, rectal irrigation, and stimulation, the temperature rose to 108.8° the following morning. The pulse was imperceptible, and the child died at 10 A.M., about nineteen hours after operation. Cultures taken from the wound immediately after death were sterile and the wound appeared all right.

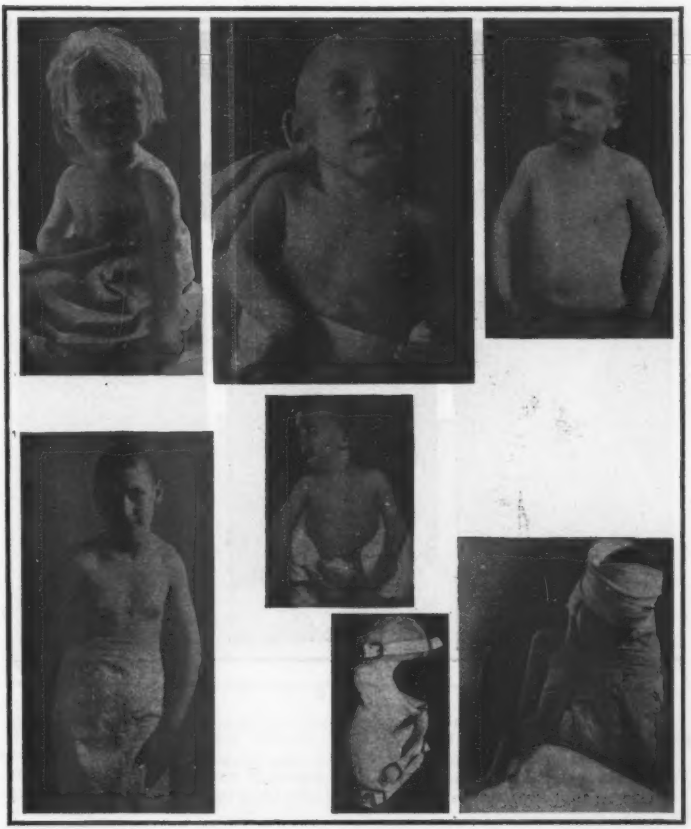
CASE II.—Annie H., aged ten years. Typical birth palsy of milder grade was present, as shown in Fig. 7, Case II., 1. The patient was a fourth child, vertex presentation, markedly asphyxiated after a dry labor of seven hours' duration. Forceps were used and great force employed in delivery. Palsy of the right arm was at once noticed. At the end of four months improvement began and continued for a year. Slight contractures have occurred in all the muscles paralyzed, producing the characteristic deformity.

Palpation disclosed a cicatricial induration, most marked at the juncture of the fifth and sixth cervical roots.

Operation June 17, 1903; chloroform. The lesser operation was done as described above (Fig. 10, 1). The deep fascia was thick-

FIG. 6

Case I., 1. Case IV., 1. Case V., 1.

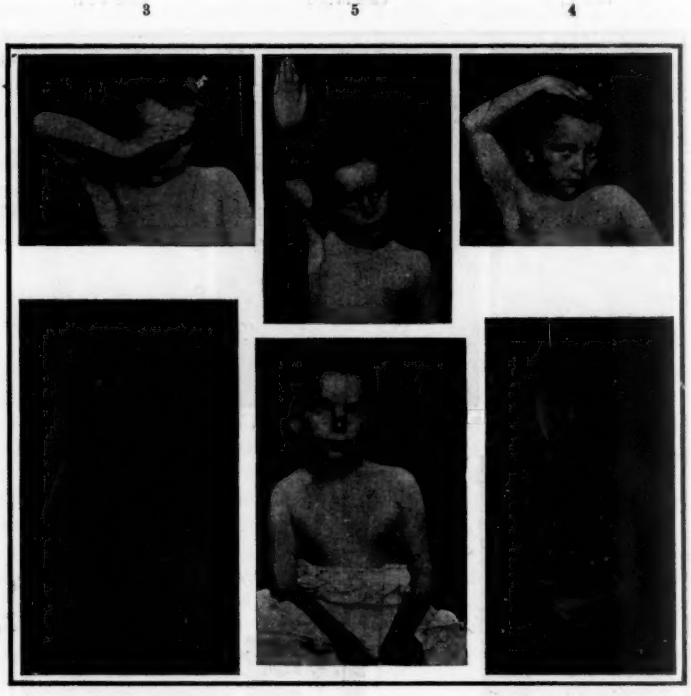


Case VI., 1. Case VII., 1 and 2. Case VI., 2.

Case I., 1. Entire brachial lesion of the left arm.
Case IV., 1. Typical laceration brachial birth palsy of the right arm.
Case V., 1. Mild degree of laceration brachial birth palsy of the right arm.
Case VI., 1. Mild degree of laceration brachial birth palsy of the left arm.
Case VI., 2. Shows the attitude maintained, without a moment's intermission, for three weeks after operation. The picture was taken at the end of three weeks.
Case VII., 1. Typical laceration brachial birth palsy of the right arm.
Case VII., 2. Shows the perfected cast for maintaining the proper postoperative position.

ened in front of the nerve lesion, which involved the fifth and sixth nerves, of which the damaged portion (about 1 cm.) was excised (Fig. 11, Case II.). The nerves were sutured and the wound closed as usual. The operation lasted one hour. Very little blood was

FIG. 7. Case II.



1. (Typical laceration brachial birth palsy.) Ordinary position in which the patient held the right arm before operation.
2. The amount of possible supination of the hand before operation.
3. Amount of elevation of arm possible before operation.
4. Showing patient's ability to place the hand on the top of the head eighteen months after operation. (Compare with 3.)
5. Showing patient's nearly normal ability to elevate the arm eighteen months after operation.
6. Showing the amount of supination of the right hand eighteen months after operation.

lost. There was no reaction. The wound healed by primary union. After three weeks the dressings were removed and the limb left free. Marked return of power began in the third month and has continued to the present time. (Compare Plate II., Case II., Figs, 1, 2, and 3 with Figs. 4, 5, and 6.) These show the improvement in range of

muscle action. The muscle power in the distribution of the fifth and sixth cervical nerve roots has been fully doubled.

CASE III.—F. G. C., aged eight years. Birth palsy of the right arm, as shown in Fig. 8, Case III., 1. The habitual position charac-

FIG. 8. Case III.

1. (Typical laceration brachial birth palsy.) Ordinary position in which patient held right arm before operation.

2. Amount of supination of the right hand possible before operation.

3. Showing extent of muscle power in the biceps and deltoid before operation.

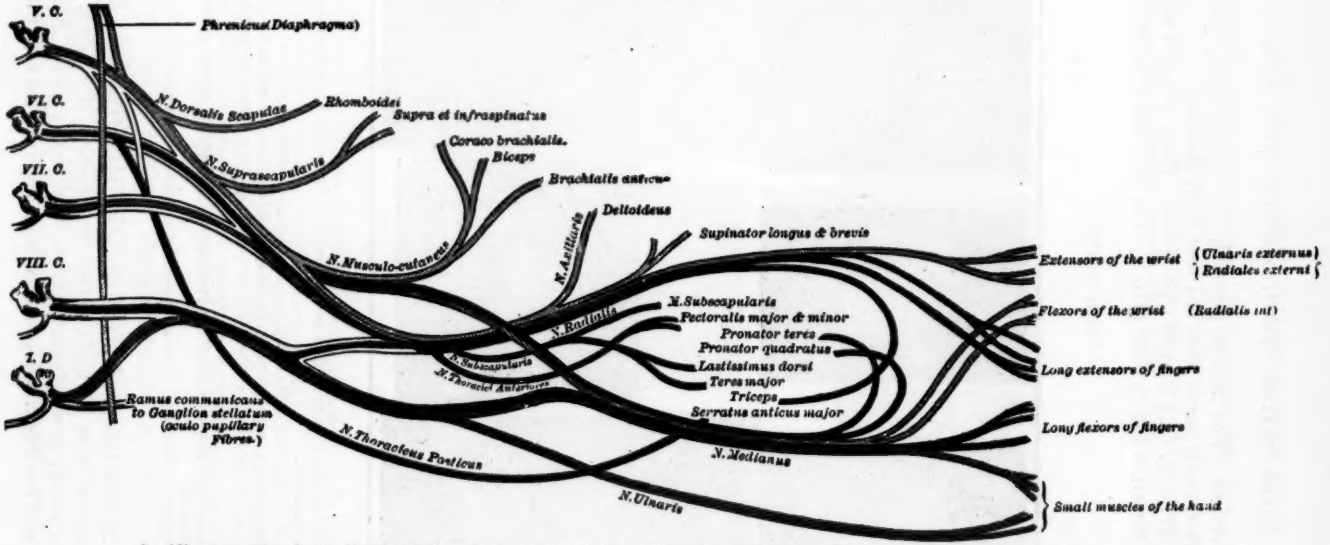
4. Showing great reduction in deformity of the right arm nine months after operation. (Compare with 1.)

5. Showing extent of muscle power in the biceps and deltoid nine months after operation. (Compare with 3.)

6. Showing still greater return of muscle power ten months after operation. (Compare with 3 and 5.)

teristic in this lesion. He was the third child (the two preceding confinements were normal), vertex presentation, dry labor, lasting fourteen hours, in which instruments were finally used. After prolonged and forcible traction he was born asphyxiated and with total

FIG. 9

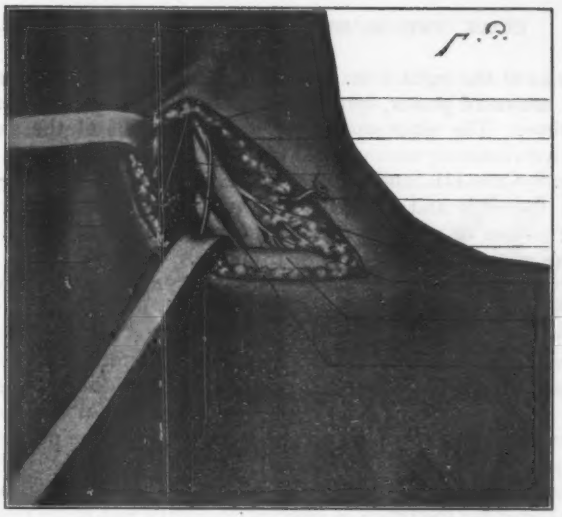


Semidiagrammatic scheme (Kocher) to show the formation of the brachial plexus and the nerve supply to the muscles of the upper extremity.

FIG. 10

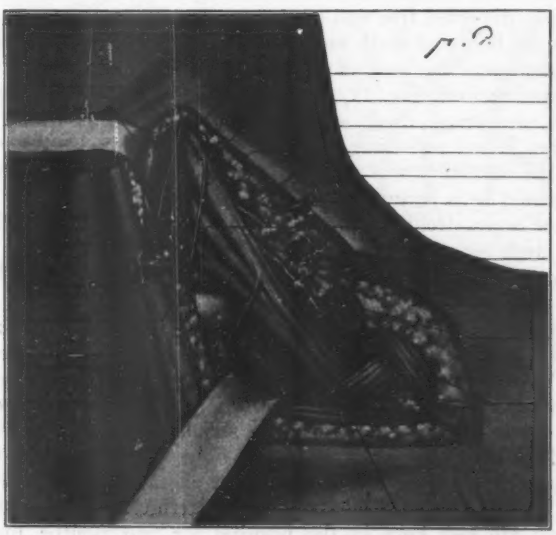
Reproductions from original drawings of dissections of the operative field.

1



1, scalenus anticus muscle; 2, phrenic nerve; 3, internal jugular vein; 4, transversalis colli artery divided; 5, VII. cervical root; 6, omohyoid muscle; 7, V. cervical root; 8, scalenus medias muscle; 9, VI. cervical root; 10, transversalis colli artery; 11, suprascapular nerve; 12, nerve to subclavius muscle; 13, clavicle; 14, nerve to scalenus anticus muscle.

2



1, phrenic nerve; 2, scalenus anticus muscle; 3, internal jugular vein; 4, transversalis colli artery; 5, omohyoid muscle, divided; 6, suprascapular artery, divided; 7, VIII. cervical and I. dorsal roots; 8, external anterior thoracic nerve; 9, subclavian artery; 10, V. cervical root; 11, VI. cervical root; 12, scalenus medius muscle; 13, nerve to scalenus anticus muscle; 14, suprascapular nerve; 15, transversalis colli artery; 16, VII. cervical root; 17, omohyoid muscle, divided; 18, suprascapular artery; 19, clavicle and subclavius muscle, divided and retracted; 20, deltoid, pectoralis minor, pectoralis major (muscles); 21, nerve to subclavius muscle.

paralysis of the right arm. During the first year there was some slight return of power, but there has been no improvement since that time. The right upper extremity and half of the shoulder girdle are distinctly undeveloped compared to the left side, as shown in Fig. 8, Case III., and the *x*-ray pictures. Palpation of the lesion shows the fifth and sixth nerves involved in a cicatricial mass. There is loss of reaction to the faradic current and R. D. in all muscles of the fifth and sixth distribution. The shoulder is a flail-joint. The elbow cannot be extended nearer than 30 degrees to a straight line. The wrist is markedly flexed and ulnar adducted. The extremity is cooler than the normal one and the hand is purple colored. The bones of the damaged extremity are from 12 to 20 per cent. shorter than the corresponding bones on the opposite side. There is a pronounced posteroinferior dislocation of the shoulder. The coracoid and acromion processes are longer, more pointed, and bend more downward and forward than normal.

Operation June 24, 1903; chloroform. The simpler procedure was resorted to; the fascia in front of the plexus was much thickened and adherent to the nerves. When the plexus was exposed, a most interesting condition was found; the external nerve division derived from the junction of the fifth and sixth cervical roots was torn away and displaced downward and inward 2.5 cm., and was adherent to the front of the scalenus anticus muscle by firm fibrous tissue. The nerve was dissected free and its damaged end removed. The junction of the fifth and sixth nerve and the posterior division derived from them was the seat of cicatricial induration. The damaged tissue was excised (Fig. 11, Case III.), the freshened ends sutured, Cargile membrane used, and the wound closed as usual. The operation lasted one hour. Primary union resulted. After three weeks the extremity was allowed free mobility. The recovery was complicated by a severe bronchitis lasting two weeks. The return of power began decidedly at the end of the fifth month. At the end of the ninth month the patient was able to move the arm as shown in Fig. 8, Case III., 5. One month later, as shown in 6. He continues to improve steadily, but slowly.

Case IV.—Joseph B., aged eight months. Typical birth palsy (Fig. 6, Case IV., 1). He was the third child. The two preceding labors were normal and easy. Vertex presentation. Labor seventy-two hours, conducted by a midwife, who, when the head was born, pulled forcibly upon it to deliver the shoulders, which were caught. Two days later the paralysis of the right arm was noticed. No improvement occurred up to the time of operation. The child was small, frail, poorly nourished, and had diarrhœa; no signs of other disease. He was kept in the hospital for two months, until his diarrhœa had disappeared and his general condition had very greatly improved.

Operation July 22, 1903. Cocaine 1 per cent. and adrenalin

1:4000. As the dissection in this case would be slight, it was thought that cocaine and adrenalin would avoid the risk of a general anæsthetic. The mixed solution was injected into the skin, which after

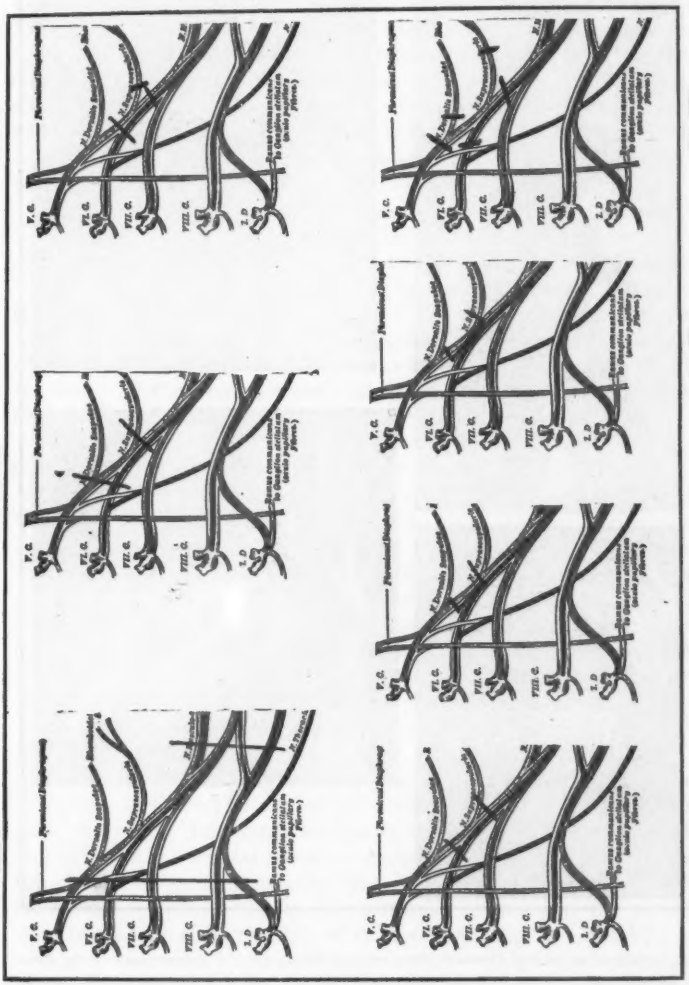


FIG. 11

Diagrams representing in a schematic way the location and amount of nerve tissue removed in each case. The portion removed lies between the black lines drawn across the nerves involved.

five minutes was divided, and the dissection carried quickly through the fat to the plexus. The cicatrix was localized at the junction of the fifth and sixth nerves and the overlying fascia. The solution

FIG. 12

1. Longitudinal section through piece excised from case: *A*, degenerated nerve fibres; *B*, hemorrhages in the perineural sheath; *C*, buckling inward of perineural sheath upon the nerve strands.

2. Band of connective tissue, *A*, lying across nerve bundle *B*.

3. Kink, *B*, in nerve fibres, *A*, which terminate in connective-tissue mass, *C*, containing pigment (blood clot).

4. Buckle in perineural sheath, *A*, which compresses the nerve fibres. Broken-up remains of the myelin sheath shown at *B*.

was injected into the fifth and sixth nerves proximally. After five minutes the cicatrix was excised (Fig. 11, Case IV.) and the operation completed as before. The operation lasted fifty minutes, and with very slight loss of blood. The child was returned to bed in fair condition, although the temperature had risen to 103°; pulse 154; respiration 80 during the operation. A few hours after the operation the diarrhœa, which had been absent for a month, recurred. The temperature remained 103° continuously and the child was very restless. In spite of treatment the temperature mounted slowly. On the third day there was suppression of urine and the child died. Cultures from the wound were sterile and there were no complications in the chest.

Case V.—George H., aged four and a half years. Second child. Vertex presentation. Very difficult labor. Instruments used after nineteen hours and "great pulling" was employed. The right arm was paralyzed at birth. Movements began to return at the end of three months, but after the end of six months improvements ceased. Paralysis, deformity, contractures, and atrophy typical of a medium grade of birth palsy, as shown in Fig. 6, Case V., 1, resulted. Electric reaction and palpation showed that the junction of the fifth and sixth nerves was the site of lesion.

Operation May 17, 1904; ether. The usual operation was done, and the fifth root from near the spine down to and including some of the junction of the fifth and sixth roots was removed (Fig. 11, Case V.). The sutures and closures were done as usual. Operation lasted thirty-five minutes. There was no reaction. Primary union resulted. The function of the muscles supplied by the fifth and part of the sixth cervical roots remained completely in abeyance for the first three months. Slow improvement has occurred since, until at the end of six months about two-thirds of the former muscle power and range of movement had returned. This child was very refractory and has followed none of the instructions as to after-treatment, and therefore the best results are not to be hoped for in his case.

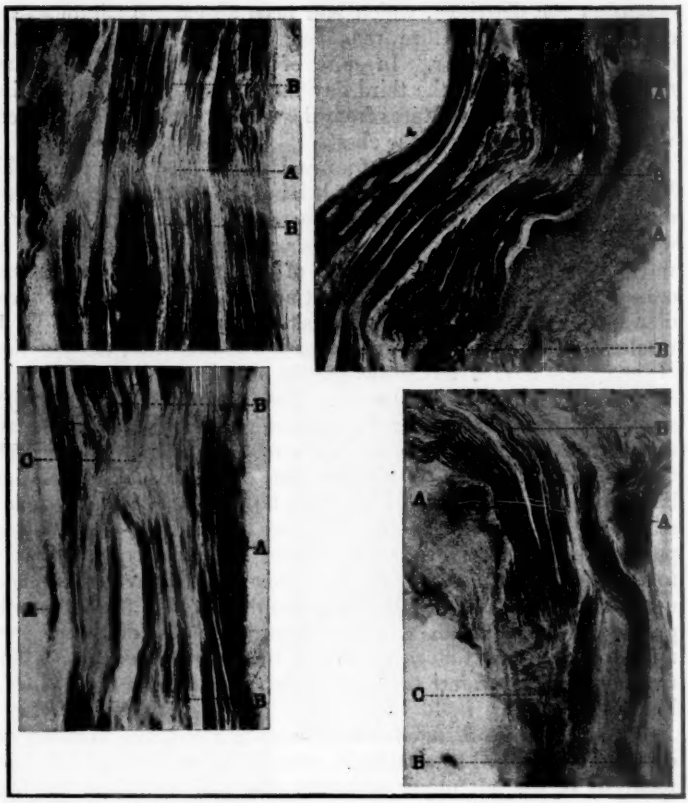
Case VI.—Rose B., aged eleven years. First child. Vertex presentation. Delivered by a midwife, who pulled on the head. Partial paralysis of the left upper extremity was noticed within the first two days. After several months improvement began and continued for about two years, appearing first in the hand muscles and then extending upward. Finally, improvement ceased and the patient now has a typical birth palsy of mild degree (Fig. 6, Case VI., 1).

The nerve lesion could not be made out by palpation in this case as in the preceding ones, but was located by the type and range of palsy at the junction of the fifth and sixth cervical root, in the upper and outer part of the fifth cervical root principally.

Operation November 12, 1904; ether. The usual operation was done. The fascia lying just in front of the nerve roots was much thickened, was adherent to the front of the scalenus anticus muscle

and to the fifth and sixth nerve roots, from all of which it was dissected away. The lesion was found in the fifth root and in the outer part of the junction of the fifth and sixth root. The damaged

FIG. 13



1. Area of compression, *A*, showing degenerated nerve fibres above and below, *B*.
2. Buckling inward of perineural sheath and pigment deposit at *A*. Torn nerve fibres, *B*.
3. An area of extreme compression: *A*, pigment deposit in perineural sheath (old hemorrhage; *B*, broken-up myelin sheath; *C*, torn and compressed nerve fibres.
4. *A*, pigment, the remains of an old hemorrhage in perineural sheath; *B*, fragments of myelin in degenerated nerve fibres; *C*, wavy bundles of torn and degenerated nerve fibres.

tissue was excised (Fig. 11, Case VI.) and the operation completed as usual. Operation lasted thirty minutes. There was no reaction and the wound healed by primary union.

In this case and the succeeding one sufficient time has not elapsed for the return of power.

CASE VII.—Edward A., aged sixteen months; fourth child. Labor was difficult; version was performed and the case was terminated as a breech. He weighed eleven pounds. Paralysis of the right arm was first noticed at the end of a week, and involved the whole extremity, which assumed the typical attitude. After about three months improvement began in the muscles of the hand and forearm, but finally ceased at fourteen months, leaving him with a typical birth palsy (Fig. 6, Case VII., 1). An induration about 1 cm. in diameter was easily palpable beneath the skin at Erb's point on the right side. He was a sturdy, well-nourished child.

Operation November 19, 1904; ether. Usual operation. The deep fascia was found thickened in front of the nerves and adherent to them. On dissecting it away carefully it was found that the fifth root had been completely torn across, proximal to its union with the sixth root. The proximal end was bulbous, displaced to and adherent to the anteroexternal part of the scalenus anticus muscle. The distal end was bulbous, was adherent to and covered the sixth root. The suprascapular nerve was fibrous for a distance of almost a centimetre from its origin. The damaged tissue was excised (Fig. 11, Case VII.), the nerve sutured, and the operation completed as usual. Operation lasted forty-five minutes. The maximum postoperative temperature was 100° (twelve hours after operation). Primary union resulted.

RESULTS (A. S. TAYLOR).

Mortality. It will be noticed that of the seven cases the first and fourth died (about 30 per cent.).

The first case was a well-developed, healthy blonde girl two years old. The operation lasted one and one-half hours; the loss of blood was moderate; all the roots of the left brachial plexus were divided at the outer edge of the scalenus anticus muscle. The clavicle was divided. This operation was of some magnitude.

The patient left the table in good condition, but within a few hours the temperature rose rapidly and steadily, as did pulse and respirations, until death occurred within twenty hours. (Temperature, 108.8°; pulse, over 200 and almost imperceptible; respirations, 68.) Cultures from the wound were sterile.

Unquestionably shock entered materially into this unfortunate result, but there was much in the clinical picture outside the realm of shock which closely identifies this case with those victims of "lymphatic diathesis," "acetonæmia," and similar little-understood conditions, in whom death occurs without reason even after trivial operations.

Infection does not enter into the problem, since the wound was dry and clean and cultures were sterile.

The fourth case was a poorly nourished child, which was in the hospital several weeks suffering from diarrhœa. During the month preceding operation there was no diarrhœa and the weight increased steadily. Immediately after the operation diarrhœa recurred in aggravated form; there was suppression of urine on the third day and the child died.

In this case death was due to the diarrhœa and the suppression of urine, but these unquestionably were precipitated by the operative procedure.

The other five cases recovered without the least evidence of shock or reaction. Even in Case VII., only sixteen months old, although the operation lasted forty-five minutes under ether anæsthesia, considerable manipulation was necessary and the fifth and sixth roots were divided, the maximum postoperative temperature was 100°, and there was no evidence of shock.

In spite of the two unfortunate cases in this series, there is every reason to believe that the mortality in a large number of cases will be very slight. Because of the complete absence of reaction in the other five cases it would seem as though the two deaths were not entirely due to the operative procedure.

Functional Improvement. The return of function in a divided and sutured nerve occurs only after months, and the longer the time elapsed between injury and operation the slower, relatively, it returns.

In cases of several years' duration there will probably never be a development of limb equal to that of its opposite fellow. This could not be expected. But the disparity (due to growth and even hypertrophy from overuse on the sound side) between the two will either not increase at all or at least will increase in a less rapid ratio.

Only in Cases II. and III. has sufficient time elapsed to give definite results. In Case II. the nutrition of the arm was good before operation. The increased range of motion is indicated in the photograph (Fig. 7, Case II.). The muscles are decidedly stronger than they were.

In Case III. the increased range of motion is shown by the photographs (Fig. 8, Case III). The extremity, instead of being cold and purplish, is now warm and of normal color. The flesh is firmer and the muscles stronger. In the shoulder, instead of a flail-joint, there is still a complete posterior dislocation, but the humerus is pulled well upward by the deltoid. The reduction of the dislocation is prevented by the distorted coracoid and acromion processes, as well as the contractures in the anterior soft structures.

He can now reach his face and head with his right hand and also button his clothing, a thing which he had never been able to do before operation.

Case V. is beginning to show return of power.

It is worthy of note that in Cases II. and III. operation was performed ten and eight years, respectively, after the injury, and yet regeneration with return of function is occurring. This is strong evidence in favor of the rule that nerve suture should be done regardless of the time elapsed since injury, provided there is muscle tissue left to be acted upon through regenerated nerve.

CONCLUSIONS.

This research embodies new facts in the etiology and pathology of the laceration type of birth palsy, the development of the relationship thereto of the symptomatology, and the establishment of a scientific basis for treatment.

1. The cause of the laceration type of birth palsy is tension on the nerve trunks, which first ruptures the nerve sheath and then the nerve fibres. The prevention of this serious lesion of the cervical nerve trunks rests with the obstetrician, who should not overstretch the child's neck in the process of delivery.

2. The persistence of the palsy is clearly explained by the pathological findings, viz.:

(*a*) Rupture of the perineural sheath with hemorrhage into its substance, resulting in the formation of hæmatomata or hæmatomatous infiltration into the neighboring tissues.

(*b*) The cicatricial contraction following organization of the blood clot and repair of the rent in the perineural sheath. The connective tissue thus formed indents and presses upon the nerve bundles, strangulating them and preventing regeneration of the nerve fibres. In some instances the same result is accomplished by the turning inward of the perineural sheath upon the nerve bundles.

3. The nature of the lesion in all cases demands excision of the damaged areas and suture of the divided ends as soon as it is proven that spontaneous repair will not take place. The plan of treatment is then the same as that for peripheral nerve injuries elsewhere.

4. In *all* cases such treatment as will prevent contractures and deformities and maintain muscle tone in the paralyzed limb should be systematically used until either spontaneous recovery occurs or operation is done. (Traumatic neuritis is a contraindication to active treatment.) It is obvious that the above measures should be continued after operation.

5. The proper time for surgical interference is not yet definitely fixed. It appears, however, to be much later than two or three months after birth, as advised in Kennedy's report. At the present time one year would seem to be a reasonable delay before operation.

6. Sufficient time has not elapsed in the majority of the cases in this series for final results to have appeared.

At the end of eighteen months in two cases (II. and III.) the improvement in nutrition, range of motion, and muscle power in the paralyzed limb have been sufficient to demonstrate the value of the operative procedure.

Bibliography.

Allard. La presse médicale, 1898, tome ii. p. 177.

Arens. Inaug. Dissert, Göttingen, 1899.

d'Astros. L'avenir des paralysies obstetricales du mémbre superieur, Rev. de méd. de l'enfance, 1892.

Budin. Bulletin médical, 1888, p. 319.

Besse. Rev. méd. de la suisse romande, 1903, tome xxiii. p. 425.

Bailly and Ominus. Arch de tocologie, 1878.

Ballenhagen. Monatsschr. f. Geburts u. Gynäkol., 1898, Bd. viii. p. 370.

Bernhardt. Zeitschr. f. klin. Med., 1882, Bd. iv. p. 420.

Bouchaud. Paralysie obstetricale d. mémbre superieur droit, Journ. d. sci. méd. de Lille, 1904, tome i. p. 49.

Barbarosa di Grovellona. Une forme particulière de paralysie obstetricale, Monoplegie brachaile double avec amyotrophie. Thèse de Paris, 1899-1900.

Clark and Taylor. Proceedings of the New York Neurological Society, Journal of Nervous and Mental Disease, 1904, vol. xcxi. p. 666.

Seeligmüller. Berl. klin. Wochenschrift, October 5, 1874.

Seeligmüller. Deutsch. Arch. f. klin. Med. 1877, vol. xx. p. 101.

Erb. Verhandl. d. Natur. Histor. med. Vereins Heidelberg (1874), 1877, p. 135.

Bernhardt. Zeitschr. f. klin. Med., 1882, Bd. iv. p. 420.

Thorburn. Medicale Chronicle, 1886, vol. iii. p. 466.

Schultz. Arch. f. Gynäkol., 1888, Bd. xxxii. p. 410.

Fieux. Annales de gynécol., 1897, tome xlvii. p. 52.

Whitman. Ibid., p. 667.

Weil. Les paralysies radiculaires obstetricales et leur traitément electrique, Journ. de physiothérapie, 1904, tome ii. p. 89.

Sechoyron. Paralysie d'origine obstétricale, Languedoc Medico-Chirurg., 1904, tome xii. p. 159.

Thoyer-Rosat. Sur la pathogénie des paralysies obstétricales, l'obstétrique, 1904, tome ix. p. 413.

Parry, R. H. Glasgow Medical Journal, 1903, vol. lix. p. 131.

Stolper. Wien. klin. Wochenschr., 1901, p. 275.

Hochstetter. Berl. klin. Wochenschr., 1893, p. 1015.

Polaillon. Arch. de tocologie, 1895, p. 245.

Helfond. Contrib. à l'etude de la paralysie radiculaires superieure, Thèse de Paris, 1896.

Guillemot. Sur le mecanisme des paralysies radiculaires obstetricales du plexus brachial, Thèse de Paris, 1898.

Weil. Revue d. mal. de l'enfance, 1896, tome xiv. p. 484.

Joly. Thèse de Paris.

Chipault. L'etat actuel de la chirurgie nerveuse, 1902, tome i. p. 377.

Henoch. Vorlesungen über Kinderkrankheiten, Berlin, 1890, 5th ed.

Mayzrier. Soc. obstét., Paris, March 21, 1901.

Docourneau. Thèse de Paris, 1876.

Oppenheimer. Lehrb. d. Nervenkr., 3te auf., 1902.

Gowers. Lancet, 1888, vol. i.

Dancher. Annal. de gynécol., September, 1901.

Carter. Boston Medical and Surgical Journal, 1892.

Glorieux. Deux cas de paralysie radiculaire brachiale totale d'origine obstétricale, La Policl., Bruxelles, August 1, 1894.

Jolly. Charité-Annalen, 1894-95, p. 21.

Lecomte. Thèse de Lille, 1894.

Peters. Vrach, 1896.

Walton. Boston Medical and Surgical Journal, 1896.

Koster. Deut. Arch. f. klin. Med., 1896, vol. lviii.

Fieux. Journ. de méd. de Bordeaux, 1896.

Haynes. Brooklyn Medical Journal, 1897.

Landolt. Ann. de gynécol., 1897.
Planchon. Lyon méd., 1898.
Peter. Annals of Gynecology and Pediatrics, 1898–99, vol. xii.
Thomas. Johns Hopkins Hospital Bulletin, 1900.
Steiner. Wien. med. Presse, 1900.
Stolper. Monatsschr. f. Geb. u. Gyn., 1901, p. 14.
Schuller. Wien. klin. Woch., 1902, p. 37.
Stransky. Centralbl. f. d. Grenzgeb. d. Med. u. Chir., 1902 (Serial).
Comby. Soc. med. des hôp., 1891, vol. vii. p. 12.
Lovett. Boston Medical and Surgical Journal, 1892, vol. xxvii. p. 8.
Burr. Ibid.
Guillemot. Ann. de gynécol., 1897, vol. xlvii. p. 35.
Cibert. Des paralysies radiculaires obstétricales au plexus brachial, Thèse de Lyon, 1897.
Doherty. Nervous Affections in Young Infants, Dublin Journal of Medical Science, 1844, vol. xxv. p. 82.
Mattei. Clinique obstétricale, Paris, 1862, 94, obs. xxiv.
Schumacher. Ueber d. Etiologie der Entbindungslahmungen, etc., Zeitschr. f. Geburts. u. Gynäkol., 1899, Bd. xli. p. 32.
Nadaud. Des paralysies obstétricales des nouveau-nes, Thèse de Paris, 1872.
Chantreuil. France méd., 1872, p. 6.
Hamond. De la paralysie des bras chez les nouveau-nes, Thèse de Paris, 1881.
Roullaud. A propos de quelques faits des paralysies des nouveau-nes, Thèse de Paris, 1887.
Kustner. Müller's Handb. d. Geburts., 1889, Bd. iii. p. 307.
Smellie. Collection of Cases and Observations in Midwifery, London, 1768, vol. ii. p. 448.
Jacquemier. Manuel des accouchements, 1846, tome ii. p. 785.
Danyon. Bull. Soc. chirurgie, Paris, 1851, tome ii. p. 148.
Guenolt. Ibid., 1867, p. 34.
Depaul. Gaz des hôpitaux, 1867, p. 90.
Duchenne. De l'electrisation localisée, 3d ed., 1872. p. 353.
Bachroch, M. H. Birth Palsies, Annals of Gynecology and Pediatrics, 1903, vol. xvi. p. 367.
Dunlop. Glasgow Medical Journal, 1903, vol. lix. p. 363.
Kennedy. Suture of the Brachial Plexus in Birth Paralysis of the Upper Extremity, British Medical Journal, February 7, 1903.
Thorburn. Journal of Obstetrics and Gynecology, 1903, vol. iii. p. 454.
Eversmann. Arch. f. Gynäkol., 1903, Bd. lxviii. p. 142.
Schuller. Jahrb. f. Psych., 1902, Bd. xxi. 397.

REVIEWS.

THE VERMIFORM APPENDIX AND ITS DISEASES. By HOWARD A. KELLY, A.B., M.D., Professor of Gynecology in the Johns Hopkins University, and E. HURDON, M.D., Assistant in Gynecology in the Johns Hopkins University, Baltimore. With 399 original illustrations, some in colors, and 3 lithographic plates. Philadelphia and London: W. B. Saunders & Co., 1905.

WHEN first glancing at this extensive work on appendicitis one is at once struck with the value of the illustrations, which are wonderfully clear and true. In fact, the illustrations tell the whole story of the appendix and its diseases, depicting its anomalies, its pathological lesions and the various methods of treating them. In no work which we have seen have the illustrations been better or so elaborate.

The work is most complete and deals with every aspect of the subject. The opening chapters have to do with the history of appendicitis and are most interesting. On reading this portion of the book one is surprised to find that long before appendicitis was generally understood and its proper treatment instituted, isolated authorities here and there over the world had described very clearly cases of appendicitis, and some of these had even recommended the proper treatment. It is evident that but for the conservatism of certain eminent authorities the advance which was made in this line of surgery after the publication of Fitz's paper in 1886 might readily have taken place nearly a generation before. In the early history of appendicitis three names stand out in this narrative: Mélier (1827), Volz (1846), and Lewis, of New York (1856). To Fitz, of Boston, however, the author gives the greatest credit.

Following the section on the history there comes the portion dealing with the anatomy of the appendix, about which it is unnecessary to say more than that it is complete and that the illustrations are deserving of especial commendation. The physiology of the appendix is given considerable space and is dealt with in a very entertaining manner. The chapters devoted to the pathology of the appendix are probably the most valuable in the book, and not the least instructive is that chapter dealing with the subject of thrombosis and its results. In this portion of the work, and also in that dealing with the symptoms and treatment, the text contains frequent brief sketches of cases which illustrate and elucidate the authors' statements. The etiology of appendicitis, which is a

matter of such general interest at the present day, is very fully and interestingly discussed. It is true that the authors are unable to furnish any altogether satisfactory explanation of its great frequency at the present day, but all the various theories in regard to this question are set forth.

The chapters on the symptoms of the various forms of appendicitis are very complete. The authors' classification of the varieties of appendicitis is, we think, very satisfactory, and its universal adoption would render reports and statistics much more comprehensive. Excellent chapters on appendicitis in typhoid fever and appendicitis in children are to be found.

The portion of the book devoted to treatment, although it may be open to the criticism of some individual surgeons, on the whole expresses the consensus of opinion of American surgeons. We are glad to see an explanation of that so frequently misunderstood term "interval operation." Kelly explains that in acute appendicitis it is never right to wait for the interval, but the interval operation should be performed when the patient has passed through an acute attack. The subject of operative technique is dealt with and all the various methods of operating for this disease and its complications fairly presented. Considerable space is given to the subject of anæsthesia, in which nitrous oxide is highly extolled.

The reference to the College of Physicians of Philadelphia as "The College of Physicians and Surgeons" in one part of the book and in another as "The College of Surgeons" is to be regretted. This is all the more noticeable because Kelly has the honor of Fellowship in this old institution.

Among the latter chapters every possible complication and sequela of appendicitis is thoroughly described. Neoplasms, specific infections, and the medicolegal aspect of appendicitis are all amply discussed.

The reader is struck throughout this exhaustive treatise with the thorough way in which the literature of the subject has been studied, and made use of, and is impressed with the enormous amount of work that this has entailed. There can be little doubt that this book contains the most complete and exhaustive study of appendicitis in all its phases that we have. J. H. G.

A Text-book of the Practice of Medicine. For Students and Practitioners. By Hobart Amory Hare, M.D., B.Sc. Philadelphia and New York: Lea Brothers & Co., 1905.

This most recent text-book of medicine promises to be as well received and as justly popular as its distinguished author's previous successful works on diagnosis and therapeutics. Dr. Hare's long

experience as a teacher and writer, combined with his extensive clinical work, has rendered him particularly able to write a book which would combine all the elements so essential for both the practitioner and student of medicine. This is especially noticeable in the sections of the work which deal with treatment. As would be expected, these are most exhaustive, interesting, and authoritative. We have never met with a medical text-book which was so thoroughly satisfactory in its method of handling the treatment of diseased conditions and in which the therapeutic side of the medical art was so well expounded. The author recognizes that the treatment of disease is, after all, the main object of the physician, and his work fully meets all requirements in this regard. In the portion dealing with the pathology and diagnosis of diseased conditions the reader is impressed with the author's clear and lucid expositions, and cannot fail to appreciate their value. One feature of the book which deserves especial commendation is the very important contribution on the subject of tropical diseases by Major Kiefer, U. S. A. There is an excellent section on diseases of the nervous system, in the preparation of which the author had the collaboration of Dr. Fickett. The illustrations with which the book abounds are well chosen and executed. The arrangement of the letter-press is such as to render the work easy of reference and to add greatly to the value of the work. J. N. H.

ON BECOMING BLIND: ADVICE FOR THE USE OF PERSONS LOSING THEIR SIGHT. By DR. EMILE JAVAL, Honorary Director of the Ophthalmic Laboratory of the Ecole des Hautes Etudes, Member of the Academy of Medicine. Translated by CARROLL E. EDSON, A.M., M.D. New York: The Macmillan Co. London: Macmillan & Co., Ltd., 1905.

THE impression left by the reading of this book is a sad one. The precepts therein contained will accomplish much to mitigate the calamity so far as may be.

Perhaps the greatest of the many misfortunes which blindness entails is loss of liberty. The central idea of Dr. Javal's work is based upon this fact; practically all the directions he has to give relate to extending the blind person's sphere of action. The home should be a model of regularity, every article should always be in its proper place so as to be readily recovered; the clock's place in the room should always be the same; its ticking will then furnish correct orientation to the sightless. The first advice to be given an adult who loses his sight is to learn Braille. In spite of its exceeding slowness, reading and writing Braille are the principal resource for hours of solitude, though the use of a special typewriter has come to replace manual writing to some extent.

The blind are particularly apt to suffer from lack of physical exercise. Home gymnastics are very tiresome. Dr. Javal highly recommends a tandem tricycle.

While opposed to marriage between blind persons, the writer does not think that marriage is necessarily contraindicated if only one of the couple is blind. The fear of blindness becoming hereditary is very slight. He concludes, however, that before marrying the blind will do well to have the subject considered twice by some devoted friends.

The book is full of useful suggestions, frequently going into minute details, but the importance of these to the blind is very great. He advises that the blind should not wear gloves, as they interfere with the sense of touch. Plates for eating may be divided in the bottom by a ridge to separate the meat from the vegetables; but to pick the bones from the fish or to put the proper amount of mustard upon the meat is nearly impossible. These acts should be done for the blind by a servant or companion. Many of the directions are simple enough, but as the writer rather pathetically remarks, why has no one thought of putting them down.

This book is not to be read for pleasure; but it is a most useful one for all who are brought in contact with the blind, most so for those who are compelled to witness this great calamity in the person of some member of their family. T. B. S.

DISEASES OF THE HEART AND AORTA. By THOMAS E. SATTERTHWAITE, M.D., Professor of Medicine in the New York Post-Graduate Medical School; Consulting Physician to the Post-Graduate, Orthopædic, and Babies' Hospital; President of the Medical Association of the Greater City of New York. New York: E. R. Pelton, 1905.

THIS book is much to be commended, for it is not too lengthy and yet presents in a remarkably clear and practical manner not only the essentials but many other original and practical points of value. The author has drawn freely on his own wide experience, and details numerous cases illustrative of particular conditions.

The chapters on syphilis of the heart and on cardiac displacements are especially interesting. The functional diseases are fully discussed, and a chapter is devoted to the consideration of Graves' disease.

Particularly valuable are the author's remarks on the general management of heart diseases, and on the Nauheim methods with American modifications. The illustrations, while not numerous, are good. F. F.

CLINICAL DIAGNOSIS AND URINALYSIS. The Medical Epitome Series. By JAMES RAE ARNEILL, A.B., M.D., Professor of Medicine and Clinical Medicine, University of Colorado, etc. Series edited by VICTOR COX PEDERSEN, A.M., M.D., Instructor in Surgery and Anæsthetist at the New York Polyclinic Medical School and Hospital, etc. Philadelphia and New York: Lea Brothers & Co., 1905.

THIS volume presents in abbreviated form the subject of laboratory diagnosis as compiled by the author from the standard textbooks and from his own observations, and it covers practically all the important points.

There is some fault to be found in the arrangement of the subject matter, especially in the opening chapters dealing with general considerations and with hæmatology. The chapters devoted to the examination of the gastric contents are excellent and entirely practical, and the same may be said of the examination of the sputum.

In the discussion of the feces the chemistry is purposely omitted. Although, as the author states, the chemical examination of the feces is usually of little value, the tests for so-called "occult" blood are certainly worthy of consideration as an aid to diagnosis in doubtful cases.

Under "Miscellaneous Examinations," exception may be taken to the brief and casual manner in which lumbar puncture is disposed of. The subject of urinalysis is covered thoroughly, and in an up-to-date manner.

The illustrations, with the exception of the colored plate supposed to represent Tallquist's color scale, are very satisfactory.

F. F.

A TREATISE ON PLAGUE: DEALING WITH THE HISTORICAL, EPIDEMIOLOGICAL, CLINICAL, THERAPEUTICAL, AND PREVENTIVE ASPECTS OF THE DISEASE. By W. J. SIMPSON, M.D. Aberd., F.R.C.P. Lond., D.P.H. Camb. Cambridge: University Press, 1905.

THE book is dedicated "to all those who are actively interested in plague and its prevention," but so engrossing is the work that one feels after reading a few chapters that the dedication must have been meant for him alone. The author's close association with the plague during the epidemic in Cape Colony and in Calcutta and Hong Kong lends a personal note to the descriptions which give the book a peculiar individuality and charm. The story of the disease, one of such great historical interest and present-day importance, is told with much accuracy and care. The first 225 pages, about one-half the treatise, are devoted to "The History and Distribution of the Plague and the Epidemics of Plague." The history

of the early epidemics, the account of the plague in India and the description of the present pandemic are given very fully, and these chapters abound with notes to literature and with quotations from the earlier writers. A very good description of the "Plague in the Individual" follows. This part is divided into several chapters on the "Morbid Anatomy and Pathology," "Channels of Infection," "Clinical Features," "Diagnosis and Prognosis," and "Treatment." Autopsy protocols and illustrative cases aid in giving an accurate idea of the disease as it is seen. Finally, the last part, covering something over 100 pages, is devoted to "Measures for Prevention and Suppression of Plague."

The book, from beginning to end, abounds in interest and points of practical importance. Maps, tables, temperature charts, and photographs help to illustrate the text. Though there are microphotographs of the plague bacillus it is rather disappointing that the author has not inserted drawings to illustrate the pathology of the disease. But one who reads this treatise cannot fail to become intensely interested in the subject. W. T. L.

WHARTON AND STILLE'S MEDICAL JURISPRUDENCE. Vol. II., Poisons. By ROBERT AMORY, A.M., M.D., and ROBERT L. EMERSON, A.B., M.D. Fifth edition. Rochester, N. Y.: The Lawyers' Co-operating Publishing Co., 1905.

THE general scope and character of this large treatise is so familiar, and the work has remained as a standard for so many years, that a detailed description is quite unnecessary. In fact, the vast amount of material which it contains almost precludes review. A very extensive and fourth revision was made in 1884, the first since 1873, and the present is the fifth edition. Much revision, of course, has been made to bring the material up to the present date, and in large part the text has been recast or rewritten. This has been done very successfully and many new subjects have been inserted, as, for example, the chapter on amyl alcohol poisoning, the discussions on poisoning by sulphonal, and the evil effects of acetanilid and the other coal-tar derivatives. Ptomain and toxalbumin poisons are also considered at some length. The newer biological tests for blood are discussed and numerous references are made to the modern work dealing with chemical problems concerned in toxicology. Occasionally one finds certain delinquencies in regard to modern methods of diagnosis, between cases of poisoning, and well-recognized diseased processes. Few criticisms, however, can be made, and, altogether, the volume is a veritable storehouse of information for both physician and lawyer. W. T. L.

THE DETECTION OF POISONS AND STRONG DRUGS, INCLUDING THE QUANTITATIVE ESTIMATION OF MEDICINAL PRINCIPLES IN CERTAIN CRUDE MATERIALS. By DR. WILHELM AUTENRIETH, Professor in the University of Frieberg. Authorized translation from the third enlarged German edition by WILLIAM H. WARREN, Ph.D., Professor of Chemistry, Medical Department of Washington University, St. Louis, Mo. Seventeen illustrations. Philadelphia: P. Blakiston's Son & Co., 1905.

THIS book is a very well-arranged and clear treatise, presenting methods for the detection of the more common poisonous substances. Although its general plan closely follows that of a manual of chemical analysis, the style is expanded sufficiently to make it easily read. The descriptions are very clear and readily understood. Several methods of qualitative and one of quantitative analysis are usually briefly given.

The various poisons are divided into three groups, and considerations of these groups constitute the first three chapters. The fourth chapter, on poisons not in the three main groups, includes a brief account of the methods of detecting poisons in blood and the biological tests for human blood. The last chapter deals with the quantitative estimation of alkaloids and other drugs in crude materials. This subject, usually omitted in American works of this kind, has apparently reached more importance in Germany, and this part of the book should be especially useful.

The book contains about 220 pages. As a laboratory guide it is sure to prove very useful. G. C. R.

THE URINE AND FECES IN DIAGNOSIS. By OTTO HENSEL, Ph.G., M.D., and RICHARD WEIL, A.M., M.D., in collaboration with SMITH ELY JELLIFFE, M.D., Ph.D. Illustrated with 116 engravings and 10 colored plates. Philadelphia and New York: Lea Brothers & Co., 1905.

LARGE text-books and treatises upon clinical diagnosis, books in which all the excretions and secretions of the body, as well as the blood, receive consideration, are numerous and well known; but it would be difficult to find a small volume covering just the ground contained in the present book. The microscopy and chemistry of the urine has been written of so often that the subject does not afford much chance for originality. The study of the feces has, however, been much neglected until quite recently, and it is with great satisfaction that one finds a book half of which is devoted exclusively to this important subject. In many obscure conditions

a careful and systematic microscopic examination of the feces clears a doubtful point or makes a diagnosis, while, as recent work has shown, no little value must be accorded to the chemical examination in investigations upon metabolism.

The first 150 pages of the book are devoted to urinalysis, and the treatment is not strikingly different from what one finds in many other text-books upon this subject. It is, however, very good, concise, and aims at the practical side of the question. In every detail the book is modern. One finds descriptions of all the newer methods of chemical examination, especially those which have been found most useful and applicable to the clinical laboratory; while older and impracticable methods handed down from text-book to text-book have wisely been omitted. Fortunately the original references are quoted. Over 150 pages are devoted to examinations of the feces. There are full chapters upon the macroscopic and microscopic examination, an excellent *résumé* on the bacteriology of the intestinal tract, taken almost entirely from Ford's work on the subject, chapters on the chemical examination with methods for the determination of total nitrogen and fat, the characteristics in disease, and the animal parasites. One might wish, perhaps, that the parasitology had been more fully discussed. Though most of the illustrations are quite good, a few are very inefficient. Such an important cut as the one representing the ova of uncinaria duodenalis ought to have been much better.

The book, however, is really excellent, and will be of great use to student and practitioner. W. T. L.

THE NAKED-EYE ANATOMY OF THE HUMAN TEETH. By THOMAS E. CONSTANT, Licentiate of the Royal College of Physicians, London; Licentiate in Dental Surgery and Member of the Royal College of Surgeons of England. Bristol: John Wright & Co. London: Simpkin, Marshall, Hamilton, Kent & Co., Ltd., 1905.

THE ignorance of medical men generally regarding the teeth is often noticed by dental surgeons and not infrequently in surgical clinics, and often causes unnecessary suffering on the part of their patients. We are inclined to believe that the medical profession would be better off for the perusal of this volume, which, however, is strictly anatomical and has nothing to say of those diseases for which the patient consults the physician and which yet have their origin in defective teeth.

This small book is especially designed for the use of dental students and fulfils its purpose admirably. Many of the illustrations are new and beautiful, but some of those relating to the anatomy of the mouth and neck are quite familiar. J. H. G.

ADDRESSES AND OTHER PAPERS. By WILLIAM W. KEEN, M.D., LL.D., F.R.C.S. Illustrated. Philadelphia and London: W. B. Saunders & Co., 1905.

DR. KEEN has put the medical profession more than ever in his debt by gathering together some of his contributions to medical literature and publishing them in book form. They cover many subjects. The first address is entitled "The Early History of Practical Anatomy." It is quite profusely illustrated with interesting and rare plates, and although not very lengthy, it constitutes a strong evidence of the erudition of the author. The second address will be read with especial interest by all the members of the profession who received their education in Philadelphia. It is a "History of the Philadelphia School of Anatomy and its Relation to Medical Teaching." This school, which flourished for over half a century, exerted a great influence on medical science in America. As Dr. Keen says, "it is only necessary to point to the thousands of men who have studied and dissected here, and here begun their scientific lives, and are now spread all over the country, and in fact all over the world, doing the best of work as practitioners, teachers, writers, and original investigators." It was not only an anatomical school; within its walls Dr. Da Costa gave private courses on physical diagnosis and did much experimental work. S. Weir Mitchell had his physiological laboratory in it, giving there the first purely experimental course on physiology ever given in Philadelphia, and in it began many of his most important physiological researches, and Dr. Hayes Agnew for many years taught anatomy.

It is a great pity that Dr. Keen's introductory delivered at the Commencement of the Woman's Medical College of Pennsylvania, entitled "Our Recent Debts to Vivisection," cannot be as universally circulated among the laity as the ridiculous literature which is disseminated by the opponents of vivisection. In this connection it is a pleasure to note that the author does reprint an article entitled "Vivisection and Brain Surgery," which appeared in *Harper's Magazine*, June, 1893. These two articles alone would make this book a valuable gift to any opponent of vivisection.

Another article which is reprinted from *Harper's Magazine* is "Recent Progress in Surgery." It illustrates Dr. Keen's happy faculty of presenting medical subjects to lay audiences which is also instanced in the article entitled "The Progress of Surgery in the Nineteenth Century," reprinted from the *New York Sun*. This article was one of a series published by that newspaper apropos of the beginning of the twentieth century, and sums up the indebtedness of the human race to the profession of surgery. There are a number of other addresses printed in the volume; all of them are of interest and value. We cannot better conclude than by again presenting our thanks to Dr. Keen and our congratulations to the medical profession on the publication. F. R. P.

A Laboratory Guide in Elementary Bacteriology. By William Dodge Frost, Ph.D., Assistant Professor of Bacteriology, University of Wisconsin. Third revised edition. New York: The Macmillan Co., 1903.

The book is designed as a guide to a course in bacteriology such as is given in the more advanced medical schools. It is composed of a well-arranged series of experiments which carry the student from the simplest technique to the more complicated bacteriological analyses. The fact that three editions have appeared in as many years shows that it has proved of use.

While some teachers undoubtedly prefer to use such a book with their classes, it seems to us that its employment perhaps detracts from the individuality of the teacher, and makes the student's work more mechanical than it might otherwise be. In planning a course of bacteriology, however, or for independent work by a novice without a teacher, the book can be highly recommended.

G. C. R.

La Rhinoplasties. Par Ch. Nelaton et L. Ombildaune. With 391 illustrations. Eight vo., 430 pages. Paris: G. Steinheil, editeur, 1904.

The volume comprises two parts, one dealing with the deformities and the other with destructive lesions of the nose, copiously illustrated with diagrams remarkably clear and easily understood. The first part treats of congenital malformations, recent and ancient traumatisms, the various operations for the relief thereof, as well as one chapter on hypertrophic acne of the nose. The destructive lesions are discussed in Part II.: first, historically; second, etiologically; and third, types—viz., (*a*) total loss of the nose; (*b*) subtotal loss (the integument and septum completely destroyed, the alæ completely or incompletely destroyed; (*c*) destruction of the base—bone and cartilage involved or destroyed.

In a very exhaustive review the following chapters give in detail the general technique of the anæsthesia, preparation of the patient, the various prosthetic apparatus, transplantations and the rhinoplastic operations depending on the employment of a pedicle of skin—or skin and bone—to replace the site of the original lesion.

The methods are exhaustively discussed and splendidly illustrated by diagrams and pictures from life (author's cases) under the headings: 1. Indian. 2. French. 3. Italian methods. The authors adopt the Indian method as including all those operations in which only a single flap of skin is used from the forehead, not including as do the Germans those methods requiring the use of a

flap from the cheeks. Under this heading the original of this operation among the Koomas as well as those of Graefe, Lisfranc, Langenbeck and others less familiar are discussed. The French and Italian methods include all the other operations recorded and give in detail the operations for the restoration of the various parts—viz., the alæ, the septum, cartilaginous, and cutaneous, as well as the various methods of support by plates of celluloid, hard rubber, metal plates, amber, etc. Under the heading of "Destruction of the Bony Skeleton" are discussed the various injection methods and apparatus, as well as an exhaustive review of the various autoplastic operations from Dieffenbach (1823) to the present day. The restoration of the tip of the nose as well as the secondary operations necessary to improve the contour of the nose after the usual contraction of the primary operation are also thoroughly gone into and discussed. A valuable feature is a criticism of the various operations at the end of each chapter and instruction as to the method to be chosen in each individual case. The work ends with a chapter on correction by means of apparatus. E. A. H.

THE PHARMACOPŒIA OF THE UNITED STATES OF AMERICA. EIGHTH DECENNIAL REVISION. By authority of the United States Pharmacopœial Convention, held at Washington, A.D., 1900. Revised by the Committee of Revision and published by the Board of Trustees. Official from September 1, 1905. Philadelphia: P. Blakiston's Son & Co.

THE new edition of the United States Pharmacopœia contains several important innovations. Probably the most striking of these is the introduction of synthetized products of definite composition which are in common use by the medical profession, the identity, purity, and strength of which are known. This does not apply to compounds of which the composition or mode of manufacture are kept secret, or which are patented. It is also to be noted that trade names are not given in the Pharmacopœia. But one serum remedy is introduced, and that is the antitoxin for diphtheria. Two animal products are introduced, namely, those made from the thyroid and suprarenal glands. Especial attention is directed by the Committee of Revision to the following changes: "The strength of tincture of aconite has been reduced from 35 per cent. to 10 per cent., and that of tincture of veratrum from 40 per cent. to 10 per cent. The strength of tincture of strophanthus has been increased from 5 per cent. to 10 per cent. These changes have been made in order to conform to the standards adopted by the International Conference on Potent Remedies held at Brussels in September, 1902, the object being to make uniform the strength of potent remedies in all parts of the world." F. R. P.

ENLARGEMENT OF THE PROSTATE: ITS HISTORY, ANATOMY, ETIOLOGY, PATHOLOGY, CLINICAL CAUSES, SYMPTOMS, DIAGNOSIS, PROGNOSIS, TREATMENT, TECHNIQUE OF OPERATION, AND AFTER-TREATMENT. By JOHN B. DEAVER, M.D., Surgeon-in-Chief to the German Hospital, Philadelphia, assisted by ASTLEY PASTON COOPER ASHHURST, M.D., Surgeon to the Out-patient Department of the Episcopal Hospital; Assistant Surgeon to the Orthopædic Hospital and to the Dispensary of the German Hospital. Illustrated with 108 full-page plates and a colored frontispiece. Philadelphia: P. Blakiston's Son & Co., 1905.

THIS book is a counterpart of that on the appendix by the same author, and from a book-making point of view is open to the same criticisms. It is light and the print is large, but the uneven and uncut leaves are a decided objection.

The first part of the work deals in an interesting manner with the History of Enlargement of the Prostate Gland. We are inclined to think that rather too much credit is given to Mr. Freyer for the origination of the present method of suprapubic prostatectomy, although no one can deny that he has done much to popularize the method. Various theories regarding the causation of enlargement of the prostate are carefully presented. The author is inclined to the view, so strongly set forth by Ciechanowski, and later confirmed by the investigations of Greene and Brooks and Crandon—viz., that a preceding inflammatory condition of the prostate is the most potent cause of enlargement.

In the section dealing with the treatment the hygienic, dietetic, and constitutional treatment are carefully outlined. Deaver believes that every patient should have a trial of catheterism before resort to prostatectomy is made, although he states plainly that the catheter can produce a cure in no case. The method of using the catheter is carefully set forth and anyone undertaking the treatment of prostatic hypertrophy by this means will do well to study this section of the book carefully. The matter is gone into in great detail, and what is said may be taken as a most reliable guide. The Bottini operation is advised in but one class of enlargement of the prostate —that in which the prostate is small and sclerosed and where its removal by prostatectomy even in otherwise healthy patients would be extremely difficult. A marked preference is shown for the suprapubic route in nearly all operations upon the prostate, either radical or palliative. Although showing this preference, Deaver states that hard, small, fibrous prostates are best removed through the perineum. He believes that the large, soft, adenomatous prostates should never be removed through the perineum. In combating the statement of those preferring the perineal route that the prostate is extravesical, one is hardly prepared to agree with the author that the prostate is no more extravesical than the appendix is extraperitoneal.

The closing chapters deal with the technique of the various methods of prostatectomy. The steps of the operation are very thoroughly and satisfactorily illustrated.

The thorough and painstaking work of Ashhurst is apparent throughout the volume. We have no hesitancy in heartily recommending this work as a guide in the treatment of all forms of enlargement of the prostate. J. H. G.

OBSESSIONS ET LA PSYCHASTHENIE. Par F. RAYMOND et PIERRE JANET. Paris: Felix Alcan, 1904.

RAYMOND and Janet have presented at great length in this volume of 537 pages a clinical and psychological study of the psychical manifestations of the fatigue neuroses. The subject-matter is presented in the form of clinical demonstrations, continuing the style made popular in France by Charcot in his Lecons du Mardi. The psychological analysis by Janet is especially thoroughly and worthy of careful attention.

Formerly the subject-matter of this nature has been treated under the subject heading of neurasthenia. The obsessions and phobias of different kinds have long since been considered as symptoms of the neurasthenic state. The cerebral type of neurasthenia, the predominance of the mental symptoms in the fatigue group, as differentiated from the spinal group or physical fatigue manifestations, has long since been abandoned by those who have given careful study to the subject. The cerebral and spinal exhaustion symptoms cannot be so differentiated, and both groups depend upon the perversion of function of the central nervous system as a whole. The psychical perturbation may dominate the clinical picture in some cases, just as the visceral introspection and physical fatigue does in others. The decrease of the will power to its almost complete abeyance, the perversion of psychical associated action, the domination of the individual by the ideas associated with this disturbance —this is the psychasthenia of Janet. The value of the separation of this group of cases from the general function group is open to question; the tagging of it with a special name complicates the subject and leads to a certain amount of confusion to the minds of those who have not given especial study to the subject. There is no question, however, of the value of the studies presented in this volume to those whose duty it is to treat such cases with an understanding of the underlying mental processes. The impression made by the volume is that the authors have made much out of little, and said it at great length. This, however, is the custom in the Charcot school to present in large and impressive volumes subject-matter which would attract more attention if a certain discrimination in case citation and a judicious elimination of unnecessary detailed discussion were attended to. D. J. McC.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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Congenital Icterus with Chronic Splenic Tumor.—v. KRANNHALS (*Deutsche Arch. f. klin. Med.*, 1904, Band lxxxi., No. 596). Minkowski in 1900 drew attention to a remarkable condition of hereditary jaundice associated with urobilinuria, splenomegaly, and siderosis of the kidneys. The instances he cites could be traced through three generations affecting at least eight members of the one family. None of the individuals observed showed any changes in the blood, while the general health was not impaired. v. Krannhals has had under his care three members of a family exhibiting similar symptoms, and from information obtained from one of these, a sister of the mother of the other two, he constructs a family tree. The grandparents of the informant had two children, a son, her uncle, who was yellow, and a daughter, her mother, who had a grayish complexion and splenic tumor; her uncle had one daughter, who was of a grayish-yellow complexion. Her mother had three daughters, of whom one died in infancy; the oldest was yellow and had a splenic tumor, as well as the informant. The oldest daughter had six children. The first pregnancy ended in an abortion. The second child had a grayish-yellow color and distended abdomen and died in infancy; the third, a boy, one of the patients, is yellow and has a splenic tumor; the fourth, a boy, who died at twenty-one years of pulmonary tuberculosis, was yellow and had a splenic tumor; the fifth, a boy, is said to be white; the sixth, a boy, one of the patients, is yellow and has a splenic tumor. The informant had one abortion and two children, who died shortly after birth, both of these being white.

In the cases under observation the stools were always colored, usually firm, never alcoholic; the urine acid, dark red in color, free from albumin, sugar, blood, and hæmoglobin; urobilin was almost constantly present, but never any bile. The blood serum showed bile. The number of red cells was below normal, 2,800,000 and 3,500,000, respectively, in the

men, and on one occasion as low as 1,000,000 in one of the women. The hæmoglobin varied from 55 to 65 per cent. The red-blood corpuscles were regular in contour, varying in size from 6 to 10μ, a few measuring as much as 12μ; there were no nucleated reds, but well-marked polychromatophilia. In at least 2 of the cases the icterus was definitely present from birth.

Cases similar to these have been reported by Hayem, Gilbert, and Bettman. In a number of the French observations the jaundice was only doubtfully congenital and the spleen not enlarged, and in some there was evidence of hepatic disease. As practically nothing is known of the changes at the bottom of the condition, the classification must remain uncertain. The only autopsy on record is taken from Minkowski on a case dead of pneumonia. No special changes were found in the liver, and the gall passages were quite clear. A small pigment stone was found in the gall-bladder. Even microscopically there were no striking changes in the liver. The intestines contained abundant bile. The spleen was enlarged, and microscopically there was simple hyperplasia and hyperæmia. The appearance of the kidneys was striking. Along the cortex there was a peculiar brownish discoloration produced by a granular pigment deposited in the epithelial cells of the convoluted tubules. Not only did this pigment give an intense reaction for iron, but there was a large amount of the metal united with proteids, which gave the reaction only after boiling with ammonium sulphide. From the ash of one kidney about 0.5 gm. of pure iron was obtained. Similar pigmentation did not occur in any other organ. Minkowski concludes that the condition depends upon some peculiar anomaly in the blood pigment transformation, perhaps consequent upon a primary change in the spleen. In Bettman's case the intensity of the jaundice displayed marked variations deepening after excesses in food and drink, after physical excitement, especially anger, after overexertion, and particularly after exposure to cold. Experimentally hæmoglobinuria could be produced by the application of cold, and Bettman assumes an abnormal tendency of the corpuscles to give off hæmoglobin to the serum, thinking that this explains best the variations in the extent of the jaundice and splenic swelling.

The Diagnostic Significance of Strangulation Marks on Round-worms.—W. EBSTEIN (*Deutsche Arch. f. klin. Med.*, 1904, Band lxxxi., p. 543) says that round-worms are remarkable for their wandering propensities, and also for their ability to force their way through very narrow openings. They have been found in the Eustachian tube and in the tear-ducts, and in an instance reported by Bruneau one had reached the external ear by way of the Eustachian tube and through a perforation in the ear drum. Not uncommonly, too, they enter the bile passages, and they have been found in the gall-bladder, and even in the ducts within the liver itself. Worms entering the common duct may not only cause obstructive jaundice, but, by carrying infectious material from the intestine, they may give rise to a suppurative inflammation of the ducts and liver, with metastatic abscesses in other organs.

Ebstein refers to a case previously reported from his clinic by Mertens. The patient, very ill with symptoms of obstructive jaundice and sepsis, began to improve immediately after the passage of two partly macerated round-worms. This striking relation of the improvement to the evacua-

tion of the worms certainly impresses one as more than a coincidence, but Ebstein urges that it establishes only a high degree of probability in favor of the worms or worm having been in the common duct. The remaining doubt, however, he thinks, is quite dispelled by the presence on one of the worms of a strangulation mark situated about 5 cm. below the head. An eminent zoölogist who examined the specimen declared that such markings never occurred normally, and was of the opinion that it could only have been produced by the worm having been struck by its front end in a small canal. This view was supported by the marked difference in diameter of the worm before and behind the constriction, a difference of 1.5 mm. Ebstein thinks that the finding of these strangulation marks may be of diagnostic importance in establishing without doubt the causative relation of round-worms to bile-duct symptoms which may have been present before their passage.

Family Tabes.—CRONER (*Berliner klin. Wochenschrift*, 1904, No. 49, p. 1266) contributes the following instances to show the relation of family to syphilitic disease of the nervous system: In a family of four children where there was apparently no hereditary taint, the three brothers all developed tabes, and the sister died at the age of fifty years of a "paralytic stroke." In another family of four brothers and two sisters, three of the men became tabetic; one brother died young of typhoid fever, one sister of diabetes, and the other sister of miliary tuberculosis. Croner thinks there must be some factors other than chance to determine the point at which the syphilitic virus strikes. Instances like the above might suggest some inherited peculiarity in the cord as an important factor in the etiology of tabes.

SURGERY.

UNDER THE CHARGE OF

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Intertesticular Anastomosis.—GATTI (*Annales des malades organes genito-urinaires*, May 15, 1905, p. 721) defines synorchidism as a fusion of the two testicles, which is an exceptional anomaly, only one case being reported, that of Geoffry St. Hilaire. The two testicles in this case had been welded along the median line, the differential lines were double, and each part of the double testicle received its own particular vessels.

Intertesticular anastomosis, as proposed by Gatti, consisted in making the excretory small canals of the interrupted testicle communicate

by way of the obstructed path, with the tubules on the internal surface of the sound testicle. In this way, through a process of degeneration, the obstructed semen could find an exit through the seminiferous tubules, the network of Haller, the epididymis and differential canal of the unaffected side.

The grafting of the path of emission of one side to that of the other side determines a degree of traumatism, to which the two testicles can respond by the phenomena of degeneration. These phenomena will be in direct proportion to the intensity of the operative traumatism, and will fail entirely when the traumatism is very slight.

When the trauma is sufficient to cause the phenomena, small diffuse zones at and near the line of anastomosis will appear, with normal zones among them, the volume of the organ remaining normal. If one of the small differential canals is delicately isolated from the connective tissue and perideferential sheath and is sectioned, it will not show the phenomena of degeneration.

When the phenomena are not too grave, there will be a proliferation in which all the tissues participate. The epithelial regeneration tends to reproduce the glandular tissue and to create a tubular anastomosis between the glandular substance of the two united organs. This takes place through slender epithelial outgrowths, at first of only one colony of cells, then more, until finally they form cylindrical plugs, which ultimately become canalized.

This epithelial regeneration is preceded and accompanied by an active vascular and nervous regeneration, which by crossing the line of anastomosis puts into communication the vascular and nervous network of the two testicles.

There will be very little proliferation of connective tissue if, without resecting any of the glandular tissue, but with incision and detachment of the tunica albuginea and the true sheaths, the surfaces to be apposed are denuded entirely with a sharp knife. The fusion of the sheath proper at the line of anastomosis must be prevented, since such a fusion would tend to separate the glandular surfaces by a proliferation of connective tissue.

Intertesticular anastomosis, as thus practised, conserves the vitality of the glandular tissue and spermatogenesis with the abundant presence of spermatozoids, and favors the normal movements. This is not only rational, but is supported by experimental proofs.

The Technique of Amputation of the Penis.—JANSEN (*Centralblatt für Chirurgie*, May 20, 1905, p. 545) calls attention to two important points demanding especial consideration. The contraction of the urinary outlet is best prevented by a ruffle-shaped formation, through the fixation of the urethral wall, which, in consequence of its unyielding character, maintains a permanent unfolding of the orifice, and more especially through a slitting of the lower wall of the urethra backward for about 1 cm. and attaching the urethra to the skin.

A greater difficulty is to form a stump, the orifice being wide enough, which will permit the escape of the urine, without staining the surrounding parts and the clothes. By the methods at present in vogue, from cicatricial contraction, the urethra turns downward and backward toward the base of the penis, so that the patients are often compelled to assume peculiar attitudes in their efforts to keep clean. The psychical

effects are disagreeable, and often sooner or later a harassing eczema of the surrounding skin sets in.

Jansen makes an oval upper flap of healthy skin, and, after ligating the dorsal artery and the veins, divides the erectile bodies transversely, leaving the urethra about 1 cm. longer. Each corpus cavernosum is closed by interrupted sutures introduced vertically, so that a horizontal line of sutures is formed, thus elevating the urethra indirectly. The projecting urethra is then turned upward over the erectile bodies and fixed by a suture, which takes hold of the albuginea of these bodies, and the periurethral connective tissue of the upper urethral wall without involving the mucous membrane. The end of the urethra is then passed through a transverse incision at the base of the long upper flap, and, after slitting the lower wall of the urethra about 1 cm. backward, its edges are sutured to the edges of the opening in the skin-flap. This flap is then turned downward, properly cut, and sutured to the skin on the under surface of the penis. Drainage must be provided for the blood which escapes under the flap, otherwise there will be a tendency to the formation of a hæmatoma. The epispadias, which is produced by the buried stitch, is lessened by the contraction of the cicatricial tissue, the scar being below, and tending to draw the urinary orifice downward. The urinary outlet will thus be finally situated on the end of the stump.

Constriction of the Ureter.—MONSARRAT (*British Medical Journal*, June 17, 1905, p. 1323) reports 3 cases of constriction of the ureter treated by operation, with good results. In the first he dilated the urethra and passed into the right ureter, through a Kelly speculum, a ureteral catheter, which stopped about three-quarters of an inch from the orifice. He then opened the abdomen and exposed the ureter transperitoneally, made a small incision into it, and passed a fine flexible bougie downward. This stopped about one inch from the bladder, and on making some pressure it was felt to be gripped in the channel, so that the ureter could be pulled upward as one can pull the urethra with an instrument tightly gripped by an urethral stricture. The bougie was then removed, the ureter closed, the peritoneum sutured over it, and the abdomen closed. Later the stricture was dilated from the bladder, when something was felt to give way, which supports the idea that the ureter was bound down in the pelvis by adhesions, being the subject of extradural and not intradural stricture.

In a second case operation showed a tumor formed of dilated kidney and pelvis, with the ureter lying on the dilated pelvis for about one and a half inches, and opening by a fine probe-sized orifice into the pelvis about three inches above the lower margin of the pelvis. A small incision was made in the outer wall of the pelvis, and a probe passed into the ureter. The intervening wall was divided and the edges thus made were sutured together on both sides of this cut. In this way the ureter was made to communicate with the pelvis by a long slit-like opening about one and a half inches in length. The final result was very satisfactory.

In the third case the constriction was at the junction of the ureter and pelvis, which was dilated. A fine pair of dressing-forceps was introduced from the pelvis and the stricture dilated. After free irrigation of the pelvis with water, the incision in the pelvis was closed and the wound in the soft parts drained. There was no return of the stricture symptoms.

Some Remarks on Cases Involving Operative Loss of Continuity of the Common Bile-duct.—MAYO (*Annals of Surgery*, July, 1905, p. 90) reports that of 1100 operations on the gall-bladder and bile passages in the hands of C. H. Mayo and himself, 159 were upon the common bile-duct, and of these 7 involved complete loss of the continuity of the common bile-duct as a direct result of the operation; 5 were intentionally produced in the attempt to remove a malignant neoplasm, 1 was caused accidentally, and 1 followed an extensive operation for gallstone disease.

His experience teaches that: first, the common duct may be united end to end by through-and-through sutures of catgut. It is essential that a few supporting sutures should be placed in the surrounding tissues, and that a portion of the circumference of the line of sutures be left open for relief of tension and drainage. Second, the common duct, and in certain cases the hepatic duct, may be implanted into the duodenum, provided a peritoneal-covered portion of the intestine be chosen for the purpose. Third, to facilitate these operations, the second portion of the duodenum should be loosened and drawn to the right and held by fixation sutures, preventing tension on the duct suture line. Fourth, drainage, if necessary, should be pliable, covered with rubber tissue and placed as distant to the suture line as will serve the purpose of protection against leakage. It is a mistake to insert gauze drainage to the suture line. The plastic lymph which should protect the suture line becomes entangled in the meshes of the gauze, and so disturbs the union as to permit, as occurred in one of Mayo's cases, a biliary fistula.

He reports a case in which, by a second operation, the hepatic duct was implanted into the duodenum about three inches from the pylorus, on account of a stricture of the common bile-duct, a result of the first operation, in which the gall-bladder had been removed. A cholecystenterostomy would have been far easier as a second operation, provided, of course, that drainage would have restored its function to a sufficient extent. Since that time Mayo has been more conservative about the removal of the gall-bladder in connection with common bile-duct surgery.

Castration for Sexual Inversion.—FERE (*Revue de chirurgie*, March, 1905, p. 293) says that castration in sexual inversion has been proposed to relieve social inconveniences, to prevent hereditary transmission, and as a cure for the inversion. The descendants of these people are certainly sometimes tainted by degeneracy, but they have in some cases given birth to perfectly normal children. Fere believes that such a radical measure is not necessary, that the tendency of inverts is not strong toward marriage, and can be still further weakened by encouraging them to lead a chaste life. Some of them are capable of conforming to the general laws of conduct. The difficulty in legalizing castration is increased if we cannot show that the operation improves the instincts of the invert, but must confess that it may make his mental condition worse. They often suffer from a consciousness of their anomaly, even to the extent of meditating suicide.

We do not know the lesion which causes the inversion. The testicle has been accused and ablation has been proposed. Fere, in one case, thought that the cause might be due to some lesion in the brain. Castration had been suggested to the patient, who showed a strong inclination to follow this advice, and consulted a neurologist, who advised against it. In a second case castration had been performed. The

testicles were replaced by two foreign bodies of about the same size and consistency as the organs. The patient believed that his aspect and expression had been changed from the sexual point of view. When the wound was healed and the dressings removed he was permitted to feel the prosthetic apparatus. He was shocked, although he had anticipated the absence of the normal sensibility of the organ. Frequent palpation led to irritation, so that it became necessary to remove the foreign bodies. He was distressed at the emptiness of the scrotum, the loss of voice, which he thought indispensable to his virility and intelligence, and the thought that everybody noticed him. He continued to be subjected to the same temptations as before the operation, and he was less capable of resisting them. The inclination to suicide which had worried him before the operation now left him, but he became addicted to the use of opium.

Fere believes that while such a case does not make an absolute rule, it deserves consideration, and should be placed with the evidence to show the history of the physical condition after castration.

Spinal Anæsthesia by Stovaine in Diseases of the Urinary Tract.—HERESCO and STROMINGER (*Annales des malades organes genitourinaires*, April 15, 1905, p. 629) made injections in 11 cases. In 10 they obtained sufficient analgesia to perform the operation. In 1, because of faulty technique, analgesia was not obtained. Their failure to make a greater number of trials is due to the fact that their results did not agree with those of other more enthusiastic observers. They had some accidents, which, although not serious enough to place the patients' lives in danger, sufficiently supported their apprehensions concerning this method.

Résumé of disturbances caused by the injections: In 2 cases the temperature was 38.3° and 38.7°. In 4 cases the temperature was 39.2°, 39.3°, 39.6°, 39.8°. In 6 cases there was slight headache; in 3 nausea; in 2 relaxation of the anal sphincter; in 1 myosis; in 3 weakness of the lower extremities; in only 1 persistent pains with contracture of the upper extremity; in 1 respiration was difficult, painful, and frequent; and in 1 there was insomnia.

Concerning the Treatment of Prostatics with Incomplete Chronic Aseptic Retention and Dilatation of the Urinary Bladder.—NICOLICH (*Annales des malades des organes genitourinaires*, May 1, 1905, p. 667) compares the unfavorable results he obtained in 10 cases of prostatic hypertrophy with retention, treated by antiseptic and successive evacuation of small quantities of urine (9 died), with his results from transvesical prostatectomy (4 cures in 4 cases).

From these results he considered that the latter method of treatment should be adopted. All 4 patients operated on had been made bedridden, probably from the use of the catheter, and had suffered severely, losing much strength. In all there was enormous retention with incontinence. One was in a very bad general condition, and another suffered from heart disease. All 4 recovered without the complications, so frequently associated with catheterization, and after from seventeen to thirty days urination was spontaneous. Such results in those cases formerly considered as *noli me tangere* cases, in the writer's opinion, makes this the treatment of first choice.

After removing the prostate he tampons the cavity with a large quantity of iodoform gauze to compress the vessels which are much dilated in a distended bladder, to ward off infection and to absorb the urine which is coming from the kidneys.

He prefers transvesical prostatectomy, because, according to his experience, it is more easy, rapid, and complete, and is less dangerous than the perineal. He compares with his own the results of M. Panchet's operations in 3 cases, in each of which there was considerable residual urine after operation. The difference is due to the superiority of the transvesical over the perineal operation.

THERAPEUTICS.

UNDER THE CHARGE OF

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Mercury in Tabes Dorsalis.—Dr. Maurice Faure states that the preferable treatment is that by injections of the soluble salts, given daily, twice or thrice a week. The injections should be intramuscular, given slowly and followed by massage. The puncture should be at least an inch and a quarter deep. The choice of the salt depends upon the case to be treated. The biniodide, the benzoate, and the bichloride are more active, but less well borne than the cacodylate or the hermophenylate when given in the same dose. In younger patients whose condition is good, the more active salts may be used in large doses. The dose, however, should be calculated for each patient according to the effect produced; when the insoluble salts are given the injection should be in one large dose per week, while the less irritating preparations are better given in repeated smaller dosage. The treatment is given in series of, in case of the insoluble salts, six to twelve injections; in case of the soluble salts, six to forty. These series are given at intervals of proper duration. The number of series in a "treatment" varies from two to four, as also does the number of "treatments" in a year. The patient should be given all the mercury that he will tolerate without exhibiting toxic symptoms, and proper diet, hygiene, and mode of life should be prescribed.—*Journal de médecine de Paris*, 1905, No. 11, p. 113.

Lumbar Puncture in Epidemic Cerebrospinal Meningitis.—Dr. L. Tobler has employed lumbar puncture as a means of treatment in 7 cases of epidemic meningitis. In 3 of these unmistakable benefit resulted. Direct relief of the severe headache was experienced; the patients rested easily, were able to sleep and take nourishment, and the stiffness of the neck and the general hyperæsthesia became less. In 1

case there was a definite fall in temperature. One of the patients proceeded directly to recovery; a second was much better in three weeks, experienced a short recurrence of the symptoms and then recovered; a third, after two punctures, was much improved, and after another puncture recovered. In the fourth case the only benefit obtained was a mitigation of the headache, and the other 3 cases did not improve after the procedure, and ended fatally.—*Correspondenz-Blatt für Schweizer Aerzte*, 1905, No. 7, p. 209.

Lumbar Puncture in Cerebrospinal Meningitis.—Prof. H. Lenhartz has treated 45 cases by this method and has found that it does no harm and frequently may be practised with much benefit. It is better not to draw off more than 8 or 10 drachms at a time. Occasionally one puncture effects a cure; at other times a number are necessary at daily or longer intervals. —*La Semaine médicale*, 1905, No. 13, p. 149.

Specific Treatment of Relapsing Fever.—Dr. Gabritschewsky has determined that this affection develops in the organism substances which are inimical to the living spirochæte, and the accumulation of these substances in the blood of the patient after each relapse has led the author toward the establishment of a serum therapy and diagnosis of this disease. Defibrinated blood from a patient is injected into the circulation of dogs or horses, these animals possessing a natural immunity to the spirochæte, and three weeks later the serum is drawn. Of 20 patients treated with this serum, 12 had but one recurrence of the fever, while 8 had two relapses, which statistics show a great advance over the ordinary treatment of the disease. In serum therapy the second relapse, when it occurs, is after a longer interval, and its duration is shorter.—*Zeitschrift für klinische Medizin*, 1905, Nos. 1 and 2, p. 43.

The Treatment of Tropical Enterocolitis.—Dr. A. Thiroux considers lavage as only a palliation and that laxatives should be used with the greatest caution, constipation, when present, being controllable by means of proper diet. Dietetic treatment is of greatest importance; a mixed diet is preferable, since a strict milk diet tends to cause constipation and flatulence, and an entirely vegetable regimen contains too little nourishment. Among other articles, the author allows fish and shellfish, meat, preferably beef, fruits, and vegetables, except cabbage; beans and peas should be divested of their outer coverings or eaten in the form of purée. The fat of meat should be removed, and butter should be eaten in small amounts only. Alcoholic drinks are not allowed, but coffee may be taken. Cold baths may be employed with benefit, and change of climate often becomes necessary. Spa treatment may at times prove useful.—*Gazette des hôpitaux*, 1905, No. 14, p. 159.

The Oat Treatment of Diabetes.—Dr. S. Lipetz believes that the good results which have been claimed for the employment of oat flour in diabetes are due to the fact that this substance, in spite of its large carbohydrate content, is not absorbed. The assertion that it is useful in combating acidosis appears to be due to the fact that any regulation of the diet increases the tolerance of carbohydrates and tends to diminish acidosis. Usually the dangers of coma can be avoided at times when acidosis is marked by giving large doses of sodium bicarbonate until

the urine becomes alkaline. The author believes it possible that in marked diabetes with alimentary disturbances the use of oat flour may lessen the tendency to intestinal fermentation, and in view of this fact, he advises the employment of small amounts of this substance in all cases.—*Zeitschrift für klinische Medizin*, 1904, Nos. 1 and 2, p. 188.

The Local Treatment of Obesity.—Dr. F. Allard considers that localized deposits of fatty tissue, such as those that occur in the thoracic or abdominal regions, are amenable to treatment by manual or vibratory massage, by exercises and by the electric current. The hand massage should be *pétrissage*, and the mechanical massage should be performed through the medium of balls or rollers. The employment of a vibrating ball controlled by an electric motor is particularly efficient. Active movements should be confined to the parts involved; the abdomen and hips are most frequently affected, and the movements recommended consist of flexion, extension, lateral movements, and circumduction of the trunk. Various apparatus may be employed, and the exercises facilitate the elimination of the excess of fat, both in the abdominal cavity and its wall; they also improve portal circulation, and consequently favorably affect the digestive function. The faradic current, through a roller electrode, of sufficient intensity to produce muscular contraction and with sufficiently frequent interruptions to tetanize the muscles, is indicated. The treatment of choice for localized fatty deposits is faradization with vibration. The metal vibrator should be attached to one pole of the battery, while a flat electrode, attached to the other pole, is applied to some other region of the body. The current should be regulated, just as when faradization alone is given.—*Revue de thérapeutique*, 1905, No. 6, p. 191.

Hydrochloric Acid in Gastritis.—Dr. F. Weitlauer states that the alkalies are inferior to hydrochloric acid in gastric catarrh. In his opinion, the dilute acid, in doses from 6 to 10 drops in water, is preferable, and acts best in combination with tannic acid in the proportion of 40 drops to 9 grains mixed with 3 ounces of water. Of this mixture a tablespoonful is taken one-half hour after the morning and evening meals in half a tumblerful of warm water. This must be rain- or distilled water, since the salts in ordinary waters are likely to neutralize the hydrochloric acid, and the dose should be taken through a tube, in order to save the teeth. The addition of the tannic acid lessens the tendency of the hydrochloric acid to disturb the stomach and exerts an astringent action upon the catarrhal condition. This treatment is indicated when the patient, several hours after a full meal, is nauseated or experiences a sense of fulness, and also in the declining stage of an acute gastritis.—*Münchener medizinische Wochenschrift*, 1905, No. 8, p. 357.

The Destiny of Carbon Dioxide in the Stomach.—Dr. K. Löning has shown by experiments upon man and animals that carbon dioxide gas taken into the stomach in water is absorbed in considerable quantity. For the first ten or fifteen minutes the absorption takes place rapidly, and after the lapse of this time more slowly until after an hour but little remains. This in alcoholic beverages also is absorbed in considerable quantity by the gastric mucous membrane.—*Zeitschrift für klinische Medizin*, 1905, Nos. 1 and 2, p. 26.

The Prevention of Puerperal Fever.—Dr. B. Credé believes in the importance of sterilizing the internal genitals after parturition. In order to accomplish this object, all clots should be removed and a suppository inserted into the uterus itself, into the cervix, or merely into the vagina. He recommends a suppository consisting of collargol and talcum powder, of each a grain and a half, cocoa butter, 30 grains. After such a suppository is inserted the vagina should be loosely packed with sterile gauze, which with the suppository may be renewed. The collargol tends to sterile the secretions without harming the tissues. If infection has taken place, douches of 1:2000 to 1:5000 collargol solution are given, with the introduction of a suppository in the intervals of these. In advanced infection the author advocates the intravenous injection of 2 to 2½ drachms of a 2 per cent. collargol solution.—*Zentralblatt für Gynäkologie*, 1905, No. 6, p. 163.

Fibrolysine.—Dr. Felix Mendel states that this combination of thiosinamine and sodium salicylate injected into the veins exercises a solvent action upon fibrous tissues, such as cicatrices, pannus, adhesions of serous membranes, etc. Direct injection into the tissues, as well as intravenous injection, is free from pain and unpleasant after-effects. Pain due to involvement of nerves in scar tissue has disappeared after a single injection of this substance into the cicatrix, but in treating such lesions the injections should be stopped as soon as the hard tissue has regained its normal texture. Further injections tend to reproduce the induration. Ankyloses due to scar contraction are especially amenable to treatment, and good results have been obtained in parametritis, which had resisted other methods. The author considers it possible that fibrolysine may have a field of usefulness in conditions of connective-tissue inflammations, such as occur in cirrhosis and nephritis.—*Therapeutische Monatshefte*, 1905, No. 2, p. 43.

Treatment of Floating Kidney.—Dr. M. I. Afanasieff considers that surgical methods are indicated in floating kidney only when the organ is the seat of malignant disease or other grave complication is present. In most cases it is possible to manage by less drastic measures. The object is to increase the fat of the patient, so that the perirenal fatty tissue may be of sufficient quantity to hold the organ in place. The diet and mode of life prescribed should be the opposite of that indicated in obesity. These means must be supplemented by the use of an abdominal belt. The kidney is replaced by manipulation and kept in position by a pad attached to the belt. This appliance is put on each morning by the patient and laced from below upward, so as to support the kidney. Medical treatment calculated to lessen the nervous state in which these patients are often found consists in the administration of belladonna, atropine, or hyoscyamus in appropriate dosage. Constipation and cardiac conditions may also require attention.—*Deutsche Medizinal-Zeitung*, 1905, No. 15, p. 157.

Diphtheria Antitoxin in Severe Whooping-cough.—Dr. Passalaequa has employed this means of treatment successfully in 7 cases. The action of the serum is usually manifest after one injection, but three or more are necessary to obtain a complete result. The diminution of the number and intensity of paroxysms is soon noticed, and while the

influence of the serum is not so rapidly shown as in diphtheria, it is, as a rule, plain by the sixth day. This form of treatment is especially indicated in cases with bronchial or pulmonary complications.—*Revue francaise de médecine et de chirurgie*, 1905, No. 11, p. 259.

Urotropine in Pyelitis.—Prof. Howard A. Kelly states that urotropine is a most valuable remedy in the prevention of inflammations of the renal pelvis. It is especially effective in cases of infection with the colon bacillus and as a prophylactic in patients with impaired kidneys, which, during the course of acute disease, are likely to become involved. —*Medical Record*, 1905, No. 14, p. 521.

Silver Iodide in Urethritis.—Drs. E. H. Siter and A. A. Uhle have used this silver salt in a number of cases with uniformly good results. In their opinion, its points of advantage are that it does not stain and is less irritating than the nitrate; it is not proprietary, and its exact strength can be controlled. Under its use the *ardor urinæ* disappears within twenty-four hours and the discharge within a week. No discomfort attends the injection of the drug in 5 to 10 per cent. strength. Silver iodide is insoluble, consequently it must be used in suspension, preferably in mucilage of quince-seed. It has also been employed in cystitis and ophthalmic conditions with good results.—*University of Pennsylvania Medical Bulletin*, 1905, No. 3, p. 89.

The Treatment of Leg Ulcers.—Dr. Vorner reports excellent results from the treatment of leg ulcers and their causative factors, eczema, and varicose veins by means of elastic roller bandages. Two are used and they should be of good length and firm; the first is snugly applied to the limb, beginning at the base of the toes, and over this the second is fitted. The proper degree of tightness can be learned by experience. The patient wears the dressing all day, but may loosen it at night, in order, if necessary, to apply other treatment. After the symptoms have disappeared the bandages should be worn for some time; then one is removed and finally the other; no recurrences of the lesions have been observed. In certain cases the immovable bandage, rendered so by a mixture of gelatin, 10 per cent.; glycerin, 5 per cent.; boric acid, 5 per cent.; gum tragacanth, ½ per cent., may be used. The mixture being applied to the outside of the first bandage and covered by the second, the ankle-joint being left free. Such a dressing may be removed by wetting with hot water.—*Münchener medizinische Wochenschrift*, 1905, No. 8, p. 350.

Local Anæsthesia in Rectal Operations.—Dr. J. R. Pennington prefers a solution consisting of beta-eucaine lactate, 3 grains; sodium chloride, 11½ grains; solution adrenalin chloride (1: 1000), 10 drops; distilled water, 3½ ounces to cocaine solutions or to sterile water injections. The solution is prepared by mixing and boiling the ingredients, except the adrenalin, which should be added when the mixture is cooled to about 100°. The entire quantity may be injected, and under its influence operations may be performed upon hemorrhoids, external, internal, thrombotic, or cutaneous polyps, prolapsus, fistula, fissure, abscesses, condylomata, etc.—*Journal of the American Medical Association*, 1905, No. 14, p. 1093.

Local Anæsthetics in Rhinolaryngology.—Dr. G. Finder considers that beta-eucaine possesses certain advantages over cocaine, in that its solutions may be boiled, keeps for a long time, and are much less toxic. He has not shown that a mixture of adrenalin and cocaine lessens the toxicity of the latter, but, with injections of ½ drachm of ½ per cent. cocaine to which 4 drops of 1:1000 adrenalin solution have been added, he has been able to do long operations upon deviated septa. In hypertrophied turbinates, where the ischæmia produced by cocaine increases the difficulty of operation, the author prefers as an anæsthetic a 1 per cent. solution of yohimbine, which does not cause vasoconstriction. Anæsthesine, in the author's opinion, lessens the dysphagia attendant upon syphilitic and tuberculous conditions of the glottis.—*Berliner klinische Wochenschrift*, 1905, No. 8, p. 211.

Formic Acid and its Salts.—Dr. H. Huchard considers these substances vigorous muscular tonics and diuretics. Sodium formiate is preferable to the acid and to other salts, and may be given in dosage of 45 to 60 grains per day, mixed with syrup of orange-peel. The author has never used the drug hypodermically. In asthenic conditions, such as are seen in convalescence from the infectious diseases, in patients affected with cardiac and arterial lesions, etc., the muscular system becomes much more vigorous and the body capable of much more exertion after the administration of the formiates, as is proven by the use of instruments calculated to measure muscular power. These substances also have a noticeable diuretic action. The urine is increased very shortly after the drug has been given and the diuresis ceases a day or two after its suspension. A point especially noteworthy concerning the effect of the medicament is that the output of urea, as well as the quantity of the urine, is markedly augmented. Long usage of the formiates does not seem to establish a tolerance, no toxic manifestations have been noted, nor is there any irritant effect upon the kidneys.—*Revue de Thérapeutique*, 1905, No. 7, p. 217.

Potassium Tellurite in Profuse Sweating.—Dr. Pochorecki has employed this substance in doses of from 0.15 to 0.75 grain daily. In the night-sweats of pulmonary tuberculosis, 0.3 to 0.5 grain, taken in the evening, prevents this unpleasant symptom, and in mild cases smaller doses are effectual. Rarely after long usage does the dose have to be increased, and while in some cases the larger doses may cause intestinal disturbance, smaller dosage is not subject to this disadvantage. The great drawback to this remedy is its alliaceous odor, but, fortunately, this is not perceived by the patient, even though it is noticeable to others, for some weeks after the cessation of its administration. The author has also employed this substance in healthy subjects who are subject to excessive perspiration in hot weather.—*Journal de médecine de Paris*, 1905, No. 15, p. 148.

The External Use of Guaiacol.—Dr. Hecht has used a 10 per cent. ointment of guaiacol and salicylic acid in acute articular rheumatism and in pleurisy with good results. Its employment has caused no unpleasant effects, even in children. In adults the effect of the treatment may not be so quickly apparent as in children, due to the fact that the skin of the former absorbs less easily. In cases of pleurisy with effusion,

many of them probably tuberculosis, the author's results have been uniformly excellent. In early cases he states that the fluid may be absorbed in from five to nine days. If the ointment causes marked irritation of the skin, a new area should be used for each application.—*Münchener medizinische Wochenschrift*, 1905, No. 9, p. 415.

PEDIATRICS.

UNDER THE CHARGE OF

LOUIS STARR, M.D.,

AND

THOMPSON S. WESTCOTT, M.D.

Prophylaxis of the Ear in Acute Infectious Diseases.—OSTMANN (*Münchener medizinische Wochenschrift*, 1905, Band lii., No. 15, p. 694) calls attention to the frequency and dangers of middle-ear complications in the course of infectious fevers, to the manner in which they arise, and to the fact that proper prophylaxis will prevent many of them. In some instances the ear affection may be laid at the door of the specific infection, but in most of the cases it is only secondary to this, the infection producing a series of local and general changes, which predispose to the ear disease. In children the Eustachian tube is relatively short and wide, and, therefore, it is easily opened during swallowing, yawning, etc.; the emaciation associated with all the acute infectious diseases removes also a certain amount of the fat of the structures of the Eustachian tube, functionally weakening its tensor muscle and leaving the tube patulous; this favors propagation of the infectious process from the throat.

This possibility of infection becomes almost a certainty in the cases where the nose and throat of the child are already the seat of chronic inflammation or of adenoid vegetations. The already existing organisms are made much more virulent by the infection, and on account of the stopped-up nose, it is comparatively easy for mucus to be pulled into the ear during forced breathing, coughing, etc. Other possibilities of setting up a middle-ear inflammation are through extension of inflammation within the tissues themselves. The nasopharyngeal catarrhs of children, therefore, should be looked after and cured; they lead to accumulation of mucus in nose and throat and prevent the child from breathing properly; the infection makes the condition considerably worse and often makes it impossible to treat it properly. The children should be accustomed at an early date to cleanse their noses and throats through proper breathing and gargling; especially unilateral, vigorous but not violent breathing should be practised. The gargling or rinsing should be performed thus: A small amount of fluid is taken in the mouth, a deep breath taken through the nose, and at the heighth of inspiration the breathing is checked; the mouth is opened, the tongue protruded, and the tongue depressed with depressor; the fluid falls into the pharynx, a choking sensation is produced and the fluid ejected;

after some practice the depressor becomes unnecessary. During the disease it is very necessary to keep the nose and throat clean; if catarrh exists a cocaine and boric solution should be used three to four times daily. The child sitting up in bed leans forward and opens the mouth; the atomizer just within the nose should have its ball compressed gently four to five times, thus cleansing not only the upper but also the posterior part of the nose; the child is then told to blow one side of the nose after the other and large amounts of mucus and pus usually come away. Renewed infection of the throat is prevented, the ear is relieved, and mixed infection is averted.

If the nose is absolutely stopped up, cocaine and adrenalin solution may have to be applied before the spray is used. The teeth and mouth must also be carefully looked after. As ear complications can arise, even after the disease process has been checked, the emaciation and weakness of the child being largely the cause of this, the prophylactic and cleansing measures should be continued for some time longer.

Cases of Sudden or Rapid Death in Pneumonia.—E. TERRIEN (*Revue mensuelle des maladies de l'enfance*, April, 1905, p. 155), referring to the rarity of sudden or rapid death in the course of or convalescence from pneumonias of infancy, quotes cases reported by Parker, Hénoch, Netter, and Viti, in which a purulent pericarditis, alone or associated with other suppurative lesions, was found at autopsy. Leroux has reported a case in a girl, aged five years, in which sudden death occurred on the third day of a pneumonia of the right apex, with hyperpyrexia. The author reports 2 cases of sudden death. In 1, an infant of eighteen months, dying on the fourteenth day, a purulent pericarditis was found, with a small collection of pus in the interlobar fissure.

The second case occurred in a girl, aged ten years, admitted to the hospital for a recent chorea. Ten days after admission the temperature rose in the evening to 101.3°, with vomiting. On the following day the temperature was the same, pulse 100, and respirations 40 to 45; there was dry, frequent cough, and a slight cyanosis of the extremities and ears. The chest showed slight dulness of the right apex, with fine rales over the upper and middle third of the lung and in the axillary region, with blowing breathing. The heart, however, showed no signs. Later in the day the child experienced an attack of suffocation, became markedly cyanotic, and died in a few minutes from asphyxia. The autopsy showed pneumonia of the right upper lobe, but there was no extrapulmonary localization, the right cavities of the heart alone showing great acute dilatation.

The following conclusions are drawn:

1. Extrapulmonary localizations are frequent, especially before the age of two years. Pericarditis, usually suppurative, is frequently encountered. It is often latent and may determine rapid death.

2. Sudden or rapid death may be observed at every age, with or without extrapulmonary localization; it is, fortunately, not a frequent accident. This possibility, in a certain degree, allies pneumonia of the child with other affections, such as diphtheria and typhoid fever, in which sudden death has been observed.

3. Cyanosis is often the first sign which reveals the imminence of a fatal termination, and may be an indication of a pericarditis or of a simple acute dilatation of the heart.

OBSTETRICS.

UNDER THE CHARGE OF

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The Mortality of Parturition in Hospital and General Practice.—In the *Journal of Obstetrics of the British Empire*, May, 1905, BOXALL publishes the results of his continued observations on the mortality of parturient patients in England, Wales, London, and the Provinces.

His statistics include death in puerperal septic disease, and also what are termed the accidents of parturition; abortion, premature labor, puerperal mania, convulsions, placenta prævia, and hemorrhage. The York Road Hospital furnishes the statistics of hospital practice. This building is an old one, but has repeatedly been altered to secure good ventilation, and for some time antiseptic precautions have been practised within the wards. In the last sixteen or more years, among 8373 patients, no deaths from septic infection occurred among those who had been for some time in the hospital, and who had not been examined by outside physicians. During this period 4 deaths took place from septic infection in patients who had been infected before admission. Septic infection from these cases did not spread to other patients in the hospital within the time of antiseptic precautions. The mortality of this hospital formerly ranged from 3.08 per cent. to 1.69 per cent. Just before antiseptic precautions were instituted, 9 patients died out of 63 delivered; as a result the hospital was closed, its ventilation remodelled, and when it was reopened a system of antiseptic precautions was instituted by a committee, of which Lister was chairman.

At present but one douche is given during the puerperal period, and that on the fifth day. Formalin has been substituted for sulphur for fumigation, and the washing of the walls and the furniture with carbolic solution has been discontinued; for a time a modern patented antiseptic salufer was used, but the results were so bad that bichloride of mercury was again employed.

From these observations, the writer concludes that the total death rate from parturition has not diminished in England, Wales, Scotland, or Ireland. In London it has declined considerably. The death rate from the accidents of parturition has declined generally, especially in London, but remains abnormally high in Ireland. For the last ten years the death rate from sepsis has declined in London; in other parts it has increased; the only explanation for this lies in the fact that it is still almost impossible to induce the general practitioner to practice antiseptic precautions in attending cases of labor.

Abdominal Hysterectomy for Severe Concealed Accidental Hemorrhage.—TARGETT (*Journal of Obstetrics of the British Empire*, May, 1905) describes the case of a multipara who went to Guy's Hospital in col-

lapse. It was stated that she had been seized with severe pain in the lower abdomen during the morning and that she had become very faint. The patient had had seven children, and with the last two there had been hemorrhage during pregnancy. During the last gestation hemorrhage occurred during the seventh month and was treated by the use of the tampon. Labor came on, the child being stillborn. The patient thought she was six months advanced in pregnancy.

When she was first seen the os uteri admitted the tips of two fingers; there had been some hemorrhage and the patient was suffering severe pain. The membranes were ruptured and a leg brought down. Some hours later the patient became pulseless; labor did not advance, and a tampon was applied and the patient removed to the hospital.

On admission she was very pale; pulse 140, very compressible. The fundus reached to the ensiform cartilage and the uterus was very tense. The legs were much swollen, and the urine contained albumin. The uterus had evidently enlarged since the patient's admission. The os uteri admitted one finger alongside the leg of the fetus, but the edge of the cervix was very tight and could not be dilated.

In view of the fact that labor had not come on after version, that the uterus had enlarged, that the patient was growing weaker, and that the cervix could not readily be dilated, it was determined to perform abdominal hysterectomy.

On opening the abdomen the uterus was almost black in color. It was brought outside the abdomen and gauze packed about the uterus. When the womb was opened a large quantity of black clotted blood was removed. The placenta was completely detached and was removed with the fetus. Hysterectomy was then performed, the stump being covered with peritoneum. Intravenous saline transfusion was performed and saline fluid left in the peritoneal cavity.

The fetus measured 40 cm. and weighed three pounds. During the first week the pulse fell slowly from 140 to 110, thickening and tenderness were present in the posterior vaginal fornix, and pus was present in the urine. This cleared up and the patient did well until the twentieth day, when she developed thrombosis of the left lower extremity. The right was also involved, but the patient finally made a good recovery.

The reason for declining the vaginal method of operation lay in the condition of the cervix, desperate state of the patient, with the evidently increasing intrauterine hemorrhage. The patient's recovery was ultimately complete.

Intraperitoneal Rupture of the Bladder.—In the *Journal of Obstetrics of the British Empire*, May, 1905, GRIMSDALE reports the case of a primipara, aged twenty-six years. She had a lingering labor under the care of a midwife, who summoned the physician when the pains became stronger. She was delivered by forceps without much difficulty. The patient stated that she had passed no urine for two days before her delivery. The nurse reported that urine had been expelled during the labor pains. The abdomen was very prominent after the patient's delivery and very tympanitic. On the following day the patient was found in collapse. She had vomited coffee-ground material. The abdomen was swollen, and the catheter brought away fluid looking like beef-tea. The patient was at once removed to a hospital and the abdomen opened. The peritoneum bulged through the wound, and when it

was excised about a gallon of blood-stained fluid gushed out. On the posterior surface of the bladder there was an area of discoloration, and near the centre an aperture through which the urine was escaping. This area was excised and the bladder closed with fine silk. The peritoneum was thoroughly flushed out and the abdominal wound closed without drainage; a glass catheter was fixed in the urethra. The patient made a good recovery.

In the literature of the subject, Walsham reports 28 cases treated by suture, of which 11 recovered. Jones has collected 54 cases treated by suture, of which 26 died and 28 recovered. In view of the high mortality the result in this case was very fortunate.

The Treatment of Eclampsia by Parathyroid Extract.—In the *Revue di clinica medica*, March 4, 1905, VASSALE reports his results in the treatment of 3 cases of eclampsia with extract from the parathyroid glands. This extract apparently causes the convulsions to cease. In the case of a child with tetany it was clearly successful.

The Permanent Results of Dilatation with Bossi's Dilator.—LICHTENSTEIN (*Archiv für Gynäkologie*, 1905, Band lxxv., Heft 1) reports the permanent results of the use of Bossi's dilator in 28 cases. In order to use this instrument successfully, anæsthesia must be profound and the patient must not struggle. The instrument must be very gradually used, and when employed in this manner no tear of the cervix necessarily results. Of the 28 cases, 6 could not be traced after leaving the hospital, 3 wrote that they were in good health, and in 18 cases an examination was made; these comprised weak pains during labor, narrow pelvis, eclampsia, and tetanus of the uterus. Among these 18 there was no tear of the cervix in 11; a very slight tear of the vaginal portion in 2; an extensive laceration, unilateral, and a bilateral tear. In 10 cases the patients were free from any bad after-effects. In 8 there were various complications. It is estimated in cases delivered by various instruments from 25 to 30 per cent. suffer considerable tears.

Incision in the Median Line to Facilitate Labor.—In place of the usual lateral incisions, FLEISCHMAN (*Wiener klin. Wochenschrift*, March 16, 1905) employs an incision in the median line of the perineum in forceps delivery, and in delayed labor from rigid perineum, where it is not necessary to use the forceps. In 60 cases the incision tore further in only 1. In all these cases it healed without complications.

Operation for Appendicitis during Pregnancy.—MAUCLAIRE (*Annales de Gynec.*, April, 1905) reports the case of a woman, aged thirty years, who had appendicitis when four months pregnant. When the abdomen was opened in the middle line turbid fluid escaped. The uterus was congested, but showed no further abnormality to complete the diagnosis. Two other incisions were made, one above the pubes, the other over the appendix. The last revealed fetid fluid, and a gangrenous appendix full of pus was removed after draining through the right lumbar region. A fourth incision was made on the left side, which let out quantities of fetid turbid fluid. Drainage tubes were introduced into the two lateral incisions. The patient recovered, and the pregnancy was not interrupted.

He also reports a second case of chronic appendicitis at the fourth month of gestation. Three months later the patient was delivered of a fetus apparently dead two months.

In discussing this paper, Segond believed that pregnant women suffering from appendicitis should be operated upon at once. In 1 case where pain was the only symptom, he found a large quantity of pus on operation; in another the appendix was gangrenous, the patient in the first month of pregnancy, with no general symptoms.

Surgical Operations during Pregnancy.—FELLNER (*Zentralblatt für Gynäkologie*, 1905, No. 18) reviews quite extensively the literature of obstetrical operations in pregnant patients. He finds that the interruption of pregnancy depends largely upon the physical condition of the mother. Where there is insufficient action of the kidneys the slightest operation is often followed by abortion; on the contrary, operations upon the external genital organs, upon the cervix uteri, the total removal of a cancerous uterus and removal of myomata, and operations for retroversions are seldom followed by interruption of pregnancy. In four-fifths of double ovariotomies pregnancy is not interrupted, even the rupture of the fetal membranes does not invariably produce interruption of pregnancy.

In all conditions where pus forms during pregnancy, whether in the middle ear or in the brain, or attended by phlebitis, or in the lungs or mediastinum, operation is directly indicated. This is true in purulent pericarditis, in phlegmon, suppuration of the gall-bladder, liver abscess, purulent gonococcus pyenephritis, and abscesses in the genital organs. Every effort must be made to prevent labor from occurring soon after the performance of an operation where pus is evacuated. This is especially true in appendicitis, which occurs not infrequently in pregnant patients. The percentage of interruptions of pregnancy in appendicitis is the same in those operated upon and those not subjected to operation. The writer gives this as from 35 to 44 per cent. The prognosis is best in these cases, and the appendix is removed as early as possible. When labor follows soon after removal, prognosis is absolutely bad. In cases where the operator does not remove the appendix, but makes an incision only to evacuate pus, the prognosis is still worse.

Malignant tumors which can be removed by operation and which occur in pregnant patients, should in all cases be extirpated. In diseases of the stomach and intestines for which operation is indicated, pregnancy should first be interrupted, and then the operation should be performed. When pregnant patients continue to do badly with nephritis, with severe lesions of the heart, in twin pregnancy and with affections of the heart and lungs which threaten life, and when retinitis appears, pregnancy should be interrupted. In cases of heart disease complicated by pregnancy, when symptoms of liver complications or failure of compensation occur, pregnancy should be interrupted.

Placental Alterations in Puerperal Eclampsia and Their Significance.—CALORNI (*Annali di ostericia*, 1905, No. 4) contributes a paper upon this subject, giving results of 2 cases of eclampsia, with microscopic examination in each case. He concludes from these investigations that the morbid phenomena of eclampsia are the expression of a most acute toxæmia, which gradually accumulates through the progressive failure

of the organs of elimination. He thinks primitive toxæmia is usually of placental origin, and secondary toxæmia of hepatic and renal derivation. He thinks that eclampsia can occur independently of hepatic or renal lesions. He draws attention to the significance of the internal secretions of the syncytial tissues in these cases. He believes that there does not exist a purely characteristic eclamptic placenta, for we may have eclampsia with a placenta entirely unaltered; in most cases, however, the syncytial zone is evidently altered. He believes that the villi of the chorion carried into the maternal circulation have also some influence in causing eclampsia. He draws attention to the resemblance of chorioepithelioma with the lesions seen in eclampsia.

The Biological Theory of Eclampsia.—In the *Zeitschrift für Geburtshülfe*, 1905, Band iv., Heft 2, LABHARDT contributes a paper in which he criticises the so-called biological theory of eclampsia advocated by Dienst and others.

From his review of the subject, he concludes that this theory has explained nothing concerning the subject. The placenta and child can in no way be considered as bodies foreign to the mother, and the absorption of their tissues cannot injure the mother's kidneys not other organs.

Cæsarean Section.—From the clinic of Würzburg DAUBER (*Zeitschrift für Geburtshülfe*, 1905, Band liv., Heft 2) reports 30 Cæsarean sections in fifteen years' time among 7533 labors, or 1 in 251. The frequency of Cæsarean section is reported by Chrobak at 1 in 548; by Braun as 1 in 402, and by Leopold as 1 in 254. 16 of these cases were celiohysterotomies and 14 were celiohysterectomies; of the latter, 6 were Porro operations and 8 were done by dropping the stump. The frequency of the conservative operation was 1 in 471 labors, and in the radical operations, 1 in 538 labors. 26 of these operations were done for contracted pelvis; 86.6 per cent. of these, 15 were rachitic, and 12 were flat rachitic pelved. One patient had had two living children by induced labor; the others had lost children in difficult labor. There were 10 cases of osteomalacia; there was 1 case of obliquely contracted pelvis; there were 3 cases of uterine myomata, which could not be pushed up out of the pelvis, and 1 case of eclampsia, where other methods of treatment had failed.

Of the 30 cases, 17 were done for the relative indication and 13 for the absolute. In choosing the form of operation, celiohysterectomy was done when it was believed that the uterus was infected; when tumors were present in the uterus; in osteomalacia; when the patient was suffering from severe disease; and when, after emptying the uterus, postpartum hemorrhage occurred which could not be checked.

In preparing patients for operation, compresses soaked in 2 per cent. formalin were bandaged upon the abdomen for twenty-four hours before operation, if possible. The abdominal surface was cleansed with hot water, soap and brush, alcohol, ether, and corrosive sublimate. The Trendelenburg posture was not employed. The abdominal incision was a large one, and no cases of hernia are reported. The uterus was turned out of the abdomen and the abdominal contents were protected by sterile compresses. In 6 cases the uterus was so large that it was thought best to open it without removing it from the abdomen, and after the removal of the child, to turn the uterus out for suture.

In 1 of these cases the patient had considerable fever after the operation. The transverse incision was employed three times; the anterior incision in other cases; in 3 of these the anterior incision was made at the fundus. The placenta was found under the anterior incision in 6 cases. No effort was made to produce adhesions between the peritoneum and the anterior uterine wall.

The uterus was closed with silk which did not pass through the decidua; two rows of catgut were then employed, one buried in the muscle, the other closing the peritoneum. The first silk sutures were tied over the other two rows. When Porro's operation was performed, a rubber tube was placed about the cervix; the peritoneum was stitched in the usual manner, needles were put through the stump, which was cauterized with Paquelin's cautery and powdered with salicylic acid and tannin. Where the stump was dropped the cervical canal was cleansed with 20 per cent. carbolic acid and peritoneum was stitched over the stump. The abdomen was closed in layers with catgut, and a subcutaneous stitch employed to complete the closure.

Hemorrhage after emptying the uterus was not alarming in any case. Ergotine was given hypodermically before the uterus was emptied.

Regarding the lives of the children, in 2 cases operation was done before the children were viable; of the remaining 28, 25 were born living; the others had perished during labor. So far as operation was concerned, there was fetal mortality.

Among the mothers there were 4 deaths; 1 from eclampsia; 1 from peritonitis, the patient having had fever before the operation; 1 from weakness on the thirty-second day, the patient having osteomalacia; and 1 dying from heart failure, having phthisis and a diseased heart before the operation. In 3 cases, then, operation may have hastened the patient's death, a mortality of 10 per cent. If the case of eclampsia be excluded, the mortality is 8 per cent. Dauber believes from these cases that the indications for the operations should be enlarged in the relative direction.

Six Cases of Cæsarean Section.—In the *American Journal of Obstetrics*, June, 1905, De Lee reports 6 cases of Cæsarean section in which mothers and children recovered. One of these sections was done because the uterus could be entered only through a fistula caused by sloughing after a previous difficult labor. The pelvis was also contracted. The uterus was removed, and the mother recovered, although she suffered from severe shock.

Other cases were those of contracted pelves, in some of whom the uterus was removed, while in others the womb was allowed to remain. In 1 case, that of a dwarf, one ovary and part of the tube were allowed to remain, although the patient was suffering at the time from gonorrhœa. As might have been expected, the patient had high fever after the operation. One case of osteomalacia was operated upon by the conservative operation, and the ovaries were removed after the uterus had been closed. In 1 patient silk employed as a suture became infected and a sinus tract resulted, which suppurated for eight months. In 1 case the patient did badly after operation until a tight abdominal binder was loosened, when the bowels moved and distention of the abdomen disappeared.

The author's record up to the time of writing has been 10 Cæsarean section, with the recovery of 9 mothers and 9 children.

GYNECOLOGY.

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Hypoplasia of the Uterus.—POLLAK (*Wiener klin. Rundschau; Zentralblatt für Gyn.*, 1905, No. 19) describes under this term the atrophy of the uterus observed in strong, overfed subjects. He regards reduction of adipose as the important point in these cases, through diet, treatment at Marienbad, etc. Intrauterine injections of hot water are highly recommended, several quarts being used at a time a few days before the expected date of menstruation. Lutein tablets are also commended.

[Since the uteri in these cases are atrophied and the os stenosed, it is difficult to see how these injections can be given easily or safely.—H. C. C.]

Pseudoascitis in Connection with Ovarian Tumors.—HOERMANN (*Münchener med. Wochenschrift*, March 21, 1905) calls attention to the fact that the free fluid, large quantities of which are often found in connection with ovarian tumors, is not a true transudate, but is really a secretion from their epithelial lining. The writer cites a case in which he operated for supposed malignant disease of the peritoneum and found that the free fluid in the cavity came from a small opening in a cystoadenoma of the ovary. Although this perforation must have existed for at least a year, symptoms of peritonitis had first appeared only four weeks before. A chemical examination of the fluid showed that its composition was identical with that contained in the cyst. In another case of ascites the opening has closed spontaneously.

The writer advises an explorative section in all cases of ovarian tumor complicated with ascites, since it is often found that the supposed malignant neoplasm is benign.

Traumatism as an Etiological Factor in Malignant Disease.—MAFFEI (*Revue prat. d'obstétrique et de gynécologie*, 1905, No. 5) concludes a study of this subject, based on several thousand cases reported by different observers, with the statement that it is exceedingly difficult to establish in any case the fact that the development of a malignant neoplasm was directly due to local lesion. He admits that this may occur, but thinks that it must be comparatively rare.

Before the surgeon decides as to the direct relation between an injury and the appearance of a tumor at this site, he should consider the questions of heredity, the condition of the part before the trauma, its exact location, character and extent, the time of appearance of the growth, and the absence of any nodule in another place which might be the original focus of disease.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

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The Question of Iridectomy in Glaucoma Simplex.—CHENEY, Boston (*Ophthalmology*, April, 1905) calls attention to the differing opinions in regard to the value of iridectomy in glaucoma simplex. Theoretically, he is in favor of it. He thinks that the question of the tension as determined by palpation is of no great importance in deciding for or against operation. The fact that a glaucomatous cup has developed is for him sufficient proof that the tension is above normal for that eye, be it supranormal, normal, or even subnormal, as compared with what we regard as normal tension. The disk cannot resist the existing intraocular pressure, whether this yielding be due to congenital or acquired want of resistance. Iridectomy in lowering the tension offers the best chance of checking the advance of the disease. Two facts deserve special consideration; one is that the eye may retain useful vision for years if not interfered with, and the other that blindness may quickly follow as a direct result of iridectomy. The patient's age may be the deciding point in many cases. As loss of sight is inevitable without operation in time, iridectomy may be advisable in comparatively young subjects, forty years of age, for example, while in persons of advanced years, the possibility of escaping blindness by death should be borne in mind. In some individuals life will become unendurable, when the prognosis of glaucoma becomes known to them. In such it may be desirable to operate, if only for its moral effect.

Treatment of Infection after Cataract Extraction.—HANSELL, Philadelphia (*Annals of Ophthalmology*, April, 1905), very justly observes that infection after cataract extraction, in spite of all known methods of asepsis, has not been altogether overcome. Statistics from operators of large experience still show that this accident occurs in from 1 to 4 per cent.; Graefe himself had reduced it to 5 per cent. years ago before asepsis was heard of.

Preventive measures include removal of conjunctival and lachrymal disease. While bacteriology is not yet able to furnish a basis as to the advisability of operation, the presence of the staphylococcus, streptococcus, gonococcus, or the diphtheria germ are positive contraindications. The pneumococcus, the diplococcus, and xerosis bacillus do not necessarily lead to purulent disease.

Endogenous sources of infection are to be removed as far as possible; these include cystitis, endometritis, kidney disease, diabetes, phthisis, rheumatism, necrosed bones, carious teeth. Skin diseases, especially

of the face, should have vigorous cleansing methods applied before operating.

Operations cannot always be done upon selected patients. Many operations are forced upon the surgeon—*e. g.*, patients suffering from chronic nephritis or diabetes, etc.

White's plan of preparing the patient for operation has shown its efficacy in the hands of various operators. In more than 500 extractions done at the Richmond Infirmary, there was only 1 case of infection, and that was mild. The method consists in the bath and purgative; the face is washed with 1: 1000 bichloride solution, and finally with alcohol. The conjunctival sac is thoroughly irrigated with 1: 10,000 and filled with bichloride vaselin (mercury bichloride, 1 gr.; sodium chloride, 5 gr.; boiled vaselin, 6 oz.). The free use of the ointment is claimed to be an effective protection against infection from without.

The preparation of the instrument and their maintenance in an aseptic condition during the entire time of the operation is, of course, a matter of supreme importance.

The signs of beginning infection resemble those of spongy iritis, from which it must be differentiated. In iritis the upper section of the cornea becomes hazy, the anterior chamber shallow, the iris swollen and hyperæmic, and the anterior chamber is partly filled with a dark mass of spongy exudation. Infection appears as a white point in the line of incision, rapidly spreading until the whole cut is a dense white line sharply outlined against the comparatively clear cornea. The anterior chamber is at first clear and later occupied by a dull white mass suspended in its upper section or protruding through the pupil. The wound may be closed or open, usually the former, because the devitalized condition of the eye and the direct cause of the infection acting in conjunction prevent closure. The edges of the incision may unite before the septic process is fairly under way, when the anterior chamber is reformed and maintains its normal depth, at least for some time.

After infection has occurred, while the outlook is bad, it is not hopeless. Local treatment consists in cauterization of the wound, subconjunctival injections and germicides inserted or injected into the anterior chamber. The cauterization is accomplished by means of the galvanocautery, silver nitrate, mercuric bichloride, carbolic acid, and the thermocautery. Darier declares that the last should not be used. He is the special advocate of subjunctival injections, the value of which, however, still remains to be determined. The insertion of iodoform in rods or disks containing up to 70 per cent. of the remedy are unquestionably of service if introduced early enough, the earlier the better. The injection of other substances than iodoform has received little attention, though bichloride, 1:1000, has given good results. Hansell reports a strikingly satisfactory case where this method was employed with the saving of useful vision.

In all cases the general treatment should be stimulating and supporting.

Peritomy for Diffuse Corneitis and Other Affections of the Cornea.—SNELL, Sheffield (*Ophthalmology*, April, 1905), from experience in over 200 cases, advocates this operation in certain diseases of the cornea; it consists in removing a band of conjunctiva, 2 to 3 mm. in width, all around the cornea; in a few cases he has practised a partial peritomy

opposite a particular portion of the cornea, which it was specially desired to affect, but the complete operation is to be preferred.

The operation may be employed as a routine treatment in diffuse corneitis, and may be expected often to materially shorten the duration of this disease with more complete recovery of transparency. It may also be used advantageously in a variety of other corneal affections, especially recurring ulcers of the cornea. The operation appears to be free from danger.

Full Correction of Myopia.—Bourgeois, Reims (*Arch. d'Ophthal.*, May, 1905), in a paper read before the Société Française d'Ophthalmologie, advocates full correction of low and moderate degrees of myopia, especially in young subjects. These form about two-thirds of the total number of myopes. For the higher grades of myopia above 12 D. there is no general rule, each case to be decided by itself. In some young subjects partial corrections gradually increased until the full is reached will often be successful.

Until about twenty years ago oculists had very generally recommended partial correction of all cases of myopia above low degrees. Since then the subject has been largely worked over by oculists of all countries. One of the most recent publications is that of Sattler, in 1904, reviewing the work of most previous writers, and recommending full correction.

Those who have practised full correction have established the fact that this method exerts a beneficial influence upon the further progress of the myopia, arresting or retarding its increase. It is accordingly especially important to prescribe such correction at the beginning, when the error is still low. This is no doubt true of the myopia caused by prolonged near work; but it is not yet proved that it also holds good in malignant or dangerous myopia; this must be treated as regards correcting glasses with great prudence.

There are two methods: full correction at once, best applied in young subjects whose myopia is of low degree, and progressive increase in the strength of the correcting lens in the higher degrees.

The writer holds that cases of myopia up to 1 D. in young subjects will do well with the full correction; when thus early discovered and corrected these cases never increase. Even up to 3 D., the full correction is well borne in young subjects. For the general application of such correction a careful examination of the eyes of all school children must be made by competent oculists. The indiscriminate sale of glasses should be regulated by law, since children who are provided with glasses for their myopia without competent examination almost always select an overcorrection, especially in low degrees of myopia.

The writer rightly holds that skiascopy is the most exact and rapid method of determining the refraction in myopia; but he is wrong, as we believe in thinking that this can be safely done without a mydriatic. If practised in this way the patient's accommodation will lead to overestimation of the refraction in many cases in young subjects. Astigmatism is also, of course, to be carefully considered in the prescription. Anisometropia must receive proper attention; the accurate adjustment of the lenses before the eyes is no less important. Where insufficiency of convergence is present, it may be compensated by decentration of the glasses.

DERMATOLOGY.

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Postexanthematic Lupus Vulgaris.—Von Veress (*Monatshefte für Praktische Dermatologie*, Band xl., No. 11), reporting a case of disseminate lupus vulgaris, observed in Unna's clinic, following varicella, declares his belief in the external inoculation theory of the origin of these cases of lupus following the exanthemata. The many lesions of the skin; the absence of metastases in internal organs; the absence of generalized emboli; the appearance of disseminate lupus after and not during the acute exanthem; the frequent increase in the eruption at a later period, all are in favor of the external origin through inoculation. The multiplicity of the lesions, the only argument in favor of the hæmatogenous origin of the affection, is equally in favor of the origin by external inoculation, since the latter may readily occur simultaneously in many places through the numerous traumata existing in the delicate skins of children.

Implication of the vessels in the structure of the lupus nodules, and the appearance of nodules in all the layers of the skin, histological features necessary to the support of the hæmatogenous theory, are wanting.

Moniliform Hairs.—Bering (*Archiv für Dermatologie und Syphilis*, Band lxxv., Heft 1) reports a marked case of moniliform hairs occurring in a boy, aged five years. The affection began soon after birth, and was accompanied by a well-defined hyperkeratosis of the follicles, not only on the scalp, but on the shoulders and extensor surfaces of the extremities. There was a distinct history of hereditary, the father, a paternal aunt, and a cousin suffering from the same disease. Microscopic examination of a portion of the diseased scalp showed the corium very firm and poor in elastic fibres. Around the vessels were clear evidences of a chronic inflammation, and here and there the glands, more particularly the sebaceous glands, were surrounded by a dense infiltrate. The sebaceous glands were few in number, dilated, and atrophic, their excretory ducts being filled with masses of cornified epithelium. In many cases the ducts were so completely closed as to entirely prevent the exit of the hair. In consequence of this obstruction to the normal growth of the hair, it was greatly twisted and distorted; and the author believes that the spindle-like enlargements upon the hairs result from mechanical causes rather than from any pathological process.

Paget's Disease of the Nipple.—TSCHLENOFF (*Dermatologische Zeitschrift*, Band xii., Heft 3, 4, and 5) believes Paget's disease must be reckoned among the cancerous affections of the skin, representing an independent form of the same. Clinically, it represents a peculiar affection of the skin which must be distinguished from eczema and other similar diseases, as well as from ordinary cancerous affections of the skin. It occurs not only in women, but in men, and attacks not only the mammary gland, but other parts of the body. Its pathological anatomy presents all the phenomena of appearances of cancer, the chief peculiarity being the presence of the peculiar cells known as Paget's cells, which are seldom seen in other affections of the skin. Those superficial diseases of the skin which precede or accompany cancerous affections are of great importance in the etiology of this malady. Pathologically, Paget's disease is a cancer from the very beginning of its development, and its division into a cancerous and an eczematous stage is of no practical advantage. The large pale cells are not, as was earlier supposed, psorospermia, but epithelial cells, which have undergone a peculiar transformation, the nature of which is not yet well understood. The parasitic origin of this malady seems probable, but remains for the future to decide. The author regards the treatment as entirely surgical.

Effect of the X-rays upon the Normal Epidermis and upon Epitheliomatous Tissues.—DALOUS and LASSERRE (*Annales de dermatologie et de syphiligraphie*, 1905, No. 4) give the results of recent histological studies in the following conclusions: The study of epithelium shows that special lesions follow the action of the *x*-ray, which the authors designate a true radioepithelitis. Analogous modifications exist in neoplastic tissues. Nevertheless, the epithelial cells are not totally destroyed by the action of the *x*-rays, certain of them appearing to be much more sensitive to the action of the rays than others. On the one hand, the cells in sound epithelium composing the generative layer and those immediately above them and the cells corresponding to these in epithelioma are much more sensitive than the prickle cells and those of the corneous layer. The spinocellular epitheliomata are less likely to be affected favorably than those of the basocellular type. These facts find their explanation in the comparative study of the elective changes which the Roentgen rays produce in certain anatomical elements of the normal epithelium.

Treatment of Inveterate Pruritus Ani.—CHARLES BALL (*British Medical Journal*, January 21, 1905) speaks in favor of surgical treatment by means of curved incisions on each side of the affected area, enclosing the whole ellipse, except in front and behind. The incisions reach down to the sphincter muscles and the flaps raised by dissection with scissors from the muscles. All bleeding must be stopped before the flaps are replaced to ensure against the formation of a hæmatoma. Retained sutures are used with intervals for drainage. These cases are referred to, showing that the operation gave relief at once and that superficial sensation may be restored without the recurrence of itching.

[This operation, it seems to us, is rarely justifiable, and for the reason that cases are seldom met with that cannot be relieved by less heroic methods.—EDS.]

Dermatitis Seborrhoica and its Relations to Alopecia and Other Conditions.—L. D. BULKLEY (*Medical Record*, May 13, 1905) reviews his experience with dermatitis seborrhoica in private and clinical practice and concludes that it is an eruption that should be recognized, since it forms about one-tenth of the cases which come to a dermatologist. Both sexes are affected in about equal proportions, and though observed at all ages from one year to eighty-nine years, but it is mainly a disease of middle life. Combining the statistics of alopecia and dermatitis seborrhoica, it appears that in 557 out of 880 cases, or 63 per cent., the loss of hair was due to the latter condition. Dermatitis seborrhoica in all its forms is regarded as more or less contagious, and that much of modern baldness is caused thereby. Its development is, however, undoubtedly favored by the various elements which lower the vitality and render the skin more susceptible to operation of local agencies. The author describes his method of treatment, which consists mainly in the application of various combinations of resorcin, sulphur, salicylic acid, carbolic acid, formalin, etc. Proper local treatment is essential, but for the best results reconstructive treatment is necessary, that the skin may not furnish such a suitable ground for the growth of the micro-organisms found in it.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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The Transport of Tetanus Toxin to the Spinal Centres through the Nerves.—TIBERTI (*Centralblatt f. Bakt. u. Parasit. Orig.*, 1905, Band xxxviii. p. 625), from a long series of experiments, draws the following conclusions regarding the transport of tetanus toxin to the nerve centres of the spinal cord. The work in the main part corroborates that of Meyer and Ransom. If one makes a subcutaneous injection of tetanus toxin in a susceptible animal, it passes for the most part into the lymph vessels and from there to the blood; in small part it is absorbed by the nerve endings and thence conducted to the nerve centres. After hypodermic injections of tetanus toxin in one leg the toxin is demonstrable with constancy in the nerve trunks of that leg. The transport of the tetanus toxin to the nerve centres does not take place by way of the lymphatics of the nerve, but through the plasma of the nerve fibres. It is, however, necessary that the axis cylinders preserve their normal integrity in order that the nerve fibres may take up the tetanus toxin and conduct it

further to the nerve centres. Along the axis cylinder the course of the toxin is solely in a cellulopetal direction. The tetanus toxin passes onward toward the nerve cells, probably because the latter exert an attraction upon the tetanus toxin with which the nerve fibres are saturated. If the toxin is injected into the muscle it spreads in the surrounding serous fluid, after which it is absorbed by the nerve endings in the muscle. The tetanus toxin inoculated in the calf muscle of a guinea-pig may be demonstrated in the corresponding sciatic nerve one and a half hours after the injection, while it appears much sooner in the blood, namely, after ten minutes. Small doses of toxin injected directly into the nerves call forth severe symptoms of tetanus, whereas, the same quantity injected subcutaneously leads to no tetanic symptoms.

When a nerve trunk is injected with toxin and a muscle innervated by the nerve likewise inoculated, the toxin seems to be hindered in its transport to the nerve centres, and no disturbances occur in that muscle region. Tetanus toxin inoculated directly into the nerves seems to have no way of reaching the nerve centres other than through the nerve fibres; for if the spinal cord is severed at a certain point the portion of the body above this section is exempt from the effects of the toxin. If a nerve trunk is severed, the muscles innervated by this nerve show none of the symptoms of tetanus when a subcutaneous injection of toxin is given. Direct inoculation of the spinal cord shortens the incubation period and gives rise to the characteristic "tetanus dolorosus." Following intravenous injections, and after a more or less prolonged period of incubation, all the muscles of the body are affected at the same time, because the toxin is absorbed synchronously by all nerve endings. In such cases the so-called local tetanus is always absent. This latter form follows subcutaneous injection or inoculation of nerves. Tetanus toxin inoculated into the circulation rapidly passes into the lymph vessels. Tetanus toxin cannot be demonstrated with any certainty in the spinal fluid.

Typhoid Meningitis.—R. I. Cole (*Johns Hopkins Hospital Reports*, 1904, vol. xii. p. 379) presents a study of meningitis as a complication of typhoid fever. Six cases have been seen in recent years at the Johns Hopkins Hospital in which meningitis occurred as a complication of typhoid fever, and in which the typhoid bacillus was isolated from the spinal fluid. From a study of these cases, and from those reported in the literature, he divides typhoid meningitides into three groups: 1. The cases of typhoid fever with meningeal symptoms, but with no demonstrable lesion or relation to the typhoid bacillus. This is the "meningism" of the French writers. 2. Cases where the typhoid bacillus can be demonstrated in the spinal fluid, but where the inflammation is of a serous character. 3. Cases of purulent meningitis due to the typhoid bacillus.

Cole reports no cases of the first group, but considers that it is probable that more careful investigation will show some of the cases to belong more properly to group two. He has collected 8 cases of serous miningitis due to the typhoid bacillus, and adds 5 cases of his own. From a study of this group of cases, the author recommends that in the study of the spinal fluid in suspected cases in the future the following procedures should be adopted: That cultures be made from large amounts, 4 to 6 c.c. of the fluid; that the pressure of the fluid as it flows

from the spinal canal be estimated, and that the cellular elements of the spinal fluid be studied. He considers that in these cases the condition is caused by toxins liberated from typhoid bacilli localized in the spinal canal.

Of the cases of the third group, that of suppurative meningitis, Cole has studied one which he presents in detail, and tabulates with 13 others collected from the literature. The clinical features of this group, as well as the foregoing, are summarized.

The author concludes that the localization of the typhoid bacilli in the meninges bears an important relationship to the cerebral and meningeal symptoms of typhoid fever. Two forms of meningitis, serous and purulent, may be produced by the typhoid bacillus, the former being, according to the author's experience, the more common. His cases well illustrate the great value of lumbar puncture in these cases, both for diagnostic and therapeutic purposes.

The study of the pathological anatomy of the case of purulent meningitis reported by Cole forms a paper by W. G. MacCallum (*Johns Hopkins Hospital Reports*, 1904, vol. xii. p. 411). He found at autopsy the usual lesions of typhoid fever, together with bronchopneumonia, acute cholecystitis, with ulceration and localized peritonitis and acute cerebrospinal meningitis. The spinal meninges were somewhat distended with a yellowish turbid fluid, from which a pure culture of typhoid bacilli was isolated. Similar fluid was found over the cerebral hemispheres.

In the exudate the microscopic study showed a large number of large mononuclear cells, which formed the most striking feature of the lesion. These cells are three to five times the diameter of a polymorphonuclear leukocyte, and are actively phagocytic. Their relative abundance is, perhaps, the only characteristic feature of the lesion, although they were found also in other forms of meningitis. The origin of these cells is discussed at some length, and the author is of the opinion that they arise from cells found in the normal meninges and especially in the walls of the veins, the chromatocytes of Ranvier, or the adventitial cells of Marchand, which Maximow brings into such close relationship with his outwandering cells or polyblasts. Thus, he considers the process almost entirely exudative, which brings the type of meningitis due to the typhoid bacillus into closer analogy with other forms of meningitis than if the chief alterations were considered as due to proliferation of the fixed tissue elements.

MacCallum also discusses the origin of the other cells of the exudate, and describes a lesion of the arteries in which the intima is lifted by collections of cells beneath it so as sometimes to occlude the vessel. This, however, is not peculiar to meningitis due to the typhoid bacillus alone.

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